This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

**Usage guidelines**

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

**About Google Book Search**

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

LANE MEDICAL LIBRARY STANFORD
1422 .J61 1891 3 STOR
Transactions of the Seventh Internationa
24503446690

LANE

MEDICAL LIBRARY

JANE LATHROP STANFORD
JEWEL FUND

م

# TRANSACTIONS

OF THE

# Seventh International Congress of Hygiene and Demography.

LONDON, AUGUST 10TH–17TH, 1891.

**Patron:—HER MAJESTY THE QUEEN.**

**President:—H.R.H. THE PRINCE OF WALES, K.G.**

## VOLUME III.

## SECTION III.

## THE RELATIONS OF THE DISEASES OF ANIMALS TO THOSE OF MAN.

EDITED BY C. E. SHELLY, M.A., M.D.,

Assisted by the HONORARY SECRETARIES of the SECTION.

LONDON:
PRINTED BY EYRE AND SPOTTISWOODE,
PRINTERS TO THE QUEEN'S MOST EXCELLENT MAJESTY.

1892.

I 61
1891
v. 3



## OFFICERS OF THE SECTION.

### PRESIDENT.

Sir NIGEL KINGSCOTE, K.C.B., Chairman of the Board of Governors of the Royal Veterinary College.

### HONORARY VICE-PRESIDENTS.

Professor ARLOING, Lyons.
Professor BANG, Copenhagen.
Professor BOLLINGER, Munich.
Dr. CARSTEN, The Hague.
Professor CHAUVEAU, Paris.
Dr. DEVAUX, Brussels.
Dr. JOHNE, Dresden.
Dr. OSTERTAG, Stuttgart.
Professor PERRONCITO, Turin.
Professor RAILLIET, Alfort.
Dr. ROUX, Paris.
Dr. D. E. SALMON, Washington, U.S.A.

### VICE-PRESIDENTS.

Professor G. T. BROWN, C.B., Director of the Veterinary Department of the Board of Agriculture.

GEORGE BUCHANAN, M.D., F.R.S., Medical Officer of the Local Government Board.

E. KLEIN, M.D., F.R.S., Lecturer on Physiology at St. Bartholomew's Hospital.

THE PRESIDENT of the Royal College of Veterinary Surgeons (Veterinary-Colonel J. DRUMMOND LAMBERT, C.B., F.R.C.V.S., Principal Veterinary Surgeon of the Army).

J. BURDON SANDERSON, M.D., F.R.S., Professor of Physiology, Oxford University.

### COUNCIL.

EDWARD BALLARD, M.D., F.R.S., Medical Department of the Local Government Board.

EDGAR CROOKSHANK, M.B., Professor of Comparative Pathology and Bacteriology at King's College.

GEORGE FLEMING, C.B., LL.D., F.R.C.V.S., late Principal of the Army Veterinary Department.

MATT. HEDLEY, F.R.C.V.S., Chief Inspector of the Veterinary Department of the Irish Privy Council.

JAMES MCCALL, F.R.C.V.S., Principal of the Veterinary College, Glasgow.

JOHN F. MCFADYEAN, M.B., B.Sc., F.R.S.E., Lecturer on Anatomy and Pathological Histology, Royal Veterinary College, Edinburgh.

SHIRLEY MURPHY, M.R.C.S., Medical Officer of Health for the County of London.

T. J. REEVES (representing the Worshipful Company of Grocers).

Professor FRED SMITH, M.R.C.V.S., F.I.C., Army Veterinary School, Aldershot.

CLEMENT STEPHENSON, F.R.C.V.S., Chief Veterinary Inspector to the County Council of Northumberland.

Sir JOHN H. THOROLD, Bart., Chairman of the Veterinary Committee of the Royal Agricultural Society of England.

GEORGE TURNER, M.B., Medical Officer of Health for East Hertfordshire.

FRANCIS VACHER, M.R.C.S., Medical Officer of Health for Birkenhead.

Sir JACOB WILSON, Governor of the Royal Veterinary College.

### SECRETARIES.

W. DUGUID, F.R.C.V.S., Assistant Inspector of the Veterinary Department of the Board of Agriculture.

G. SIMS WOODHEAD, M.D., F.R.S.E., Director of the Research Laboratories of the Conjoint Board of Royal College of Physicians and Royal College of Surgeons.

ERNEST CLARKE, F.S.A., Secretary of the Royal Agricultural Society of England.



38705

## Note.

As was to be expected, Section III. (The Relations of the Diseases of Animals to those of Man) and Section II. (Bacteriology) to some extent overlap,—insomuch as each is concerned with certain subjects which may be regarded as more or less common to both Sections, while belonging exclusively to neither. Of such subjects, Rabies and Tuberculosis are conspicuous examples, and papers under each of these headings were contributed to both Sections. In order, therefore, to avoid needless repetition or discontinuity, as well as to make reference easier, it has been decided to group all the papers on Tuberculosis in Volume II. and all those on Rabies in Volume III. Appropriate notes and cross references will serve to indicate the chronological position assigned to each paper in the proceedings of that Section to which it was originally contributed.

# TABLE OF CONTENTS.


| | Page. |
|---|---|
| Presidential Address | 7 |
| Prévention de la Rage après Morsure.—Résultats obtenus à l'Institut Pasteur au moyen des inoculations antirabiques | 8 |
| The Propagation and Prevention of Rabies | 16 |
| *Études sur la Rage et sur la Vaccination Antirabique | 22 |
| *Ueber die praktischen Erfolge der antirabischen Schutzimpfungen in Budapest, während des ersten, vom 15 April 1890—14 April 1891, sich erstreckenden Jahres | 30 |
| *Résultats statistiques de l'Institut Antirabique d'Odessa | 35 |
| *Sur le Traitement Antirabique de Pasteur | 42 |
| *Les Résultats des Vaccinations Antirabiques à St. Pétersbourg depuis le 1–13 Juillet 1886 au 1–13 Janvier 1891 | 46 |
| *Résultats statistiques de l'Institut Antirabique de Tiflis | 47 |
| *Les Résultats des Vaccinations Antirabiques obtenus à Varsovie | 51 |
| Discussion | 53 |
| Trichinosis in the Netherlands | 56 |
| Les Parasites transmissibles des Animaux à l'Homme, envisagés spécialement au point de vue de la Prophylaxie | 57 |
| Sur les Végétaux Parasites (non microbiens) transmissibles des Animaux à l'Homme et réciproquement | 88 |
| Discussion | 100 |
| On Meat Infections—Food Poisoning | 102 |
| The Infection of Meat and Milk | 118 |
| Discussion | 127 |
| Infectious Udder Diseases of the Cow in relation to Epidemic Diseases in the Human Subject | 130 |
| Discussion | 148 |
| Die Regelung der Milchversorgung mit Hinsicht auf übertragbare Krankheiten | 155 |
| Discussion | 166 |
| The Inspection of Meat with reference to the Spread of Disease | 171 |
| The Inspection of Meat with regard to the Prevention of Disease | 177 |
| Discussion | 185 |
| Maladies Infectieuses des Animaux qui peuvent être transmises à l'Homme et réciproquement | 189 |
| Actinomycosis | 192 |
| Trois Cas d'Actinomycose chez l'Homme | 197 |
| Discussion | 199 |
| Sur les Dangers que le Charbon (Anthrax) fait courir aux Ouvriers des différents Corps de Métiers | 203 |
| Anthrax and its Relations to Workers in various Trades | 211 |
| Discussion | 218 |
| Veterinary Hygiene | 221 |
| Discussion | 246 |

INDEX.


* Originally contributed to SECTION II.

# SECTION III.

# THE RELATIONS OF THE DISEASES OF ANIMALS TO THOSE OF MAN.

## Tuesday, 11th August, 1891.

The President, SIR NIGEL KINGSCOTE, K.C.B., in the Chair.

Vice-Presidents of the day:
Professor PERRONCITO, Turin;
Veterinary-Colonel J. D. LAMBERT, C.B., President of the Royal College of Veterinary Surgeons.

## Presidential Address

BY

SIR NIGEL KINGSCOTE, K.C.B.

My first duty, in taking the Chair of this very important and interesting Section of the First International Congress of Hygiene held in the United Kingdom, is to offer to you all, on behalf of the Organising Committee and the Council of the Section, a cordial welcome to our meetings, and to express to you our sincere thanks for your presence and co-operation in the work that is before us. The subjects that we shall have to discuss are of the greatest and most immediate importance to all classes of the community in every country; and I think myself happy in having been honoured with the presidency of the first assemblage of medical men, veterinarians, and agriculturists that has ever met on common ground to discuss questions of the profoundest interest to them and to the world at large.

The title of our Section is sufficiently indicative of its scope and objects. The Relations of the Diseases of Animals to those of Man have been the subject of more or less vague speculation by observers from an early period of the world's history; but it is only of late years that such relations have been systematically and scientifically studied. It would be in the highest degree inappropriate for me, who owe my position here to the accident of being somewhat intimately associated with the business management of two important institutions which devote much of their energies to the study of animal life and animal diseases, to attempt to summarise the stages of development of this great question, or to pass in review the various diseases which have been shown to be intercommunicable between men and animals. As to some of these, rabies and anthrax, for instance, the evidence appears absolutely conclusive. A few others are, perhaps, still within the range of speculation, or at least can

hardly claim to have been proved to demonstration. Of all these diseases and disorders, and the consequences which they involve, we are fortunate in having some of the highest living authorities here to instruct us; and I shall not abuse your good nature by standing between you and the study of their researches and experience.

There can be no doubt that the anatomical and physiological relations between the lower animals and the human being are sufficiently close to justify the presumption that they are liable to the same class of diseases; and one important part of the subject which will be considered during the meetings of our Section is the question of susceptibility, without which it would seem that the individual ceases to be responsive to the attack of the malignant virus.

In short, as will probably appear in a very prominent form in the discussion of the papers on parasitic diseases, the whole question seems to resolve itself into the Survival of the Fittest; and it must be for this Congress to consider and discuss the best ways of combating the invasions of the human body by the many varieties of self-multiplying alien germs and external morbific influences which prevent the individual life from completing its normal course.

## Prévention de la rage après morsure. — Résultats obtenus a l'Institut Pasteur au moyen des inoculations anti-rabiques.

PAR

le Dr. E. Roux, de l'Institut Pasteur, Paris.

Il y a un peu plus de six ans (le 6 Juillet 1885), qu'au laboratoire de M. Pasteur était pratiquée pour la première fois sur l'homme l'inoculation préventive contre la rage. Depuis, des milliers de personnes, mordues par des animaux enragés, sont venues réclamer le traitement anti-rabique, et pour leur donner des soins il a fallu installer un service spécial qui a été l'origine de l'Institut Pasteur. Au précédent congrès d'hygiène, réuni à Vienne en 1887, mon collègue M. Chamberland exposait ce que la découverte encore nouvelle de M. Pasteur avait déjà donné, et vous vous rappelez sans doute qu'à cette époque les contradicteurs ne manquaient pas. Aujourd'hui, un assez grand nombre de personnes ont été traitées, et un temps assez long s'est écoulé pour que l'on puisse examiner les résultats de cette large pratique, et porter un jugement sur la méthode Pastorienne de prévention de la rage après morsure. Bien que des documents circonstanciés aient été publiés sur le sujet dans les Annales de l'Institut Pasteur, j'ai pensé qu'il y aurait intérêt à jeter un coup d'œil d'ensemble sur la statistique des six années qui viennent de s'écouler.

L'application de la vaccination anti-rabique à l'homme mordu, n'était admissible qu'autant qu'il était prouvé, par des expériences

irréprochables, que des animaux tels que les chiens, inoculés de la rage pouvaient ensuite être mis, par ce moyen à l'abri de la maladie. M. Pasteur a donné maintefois cette preuve avant d'essayer sur l'homme le traitement préventif. Ce traitement est donc fondé sur des faits bien établis que chacun peut reproduire avec facilité, et vous savez tous, Messieurs, que leur exactitude a été vérifiée, ici même, à Londres, par une commission de savants éminents; depuis, ils n'ont plus été contestés.

Ainsi qu'il l'avait fait tour-à-tour pour le choléra des poules, le charbon et le rouget des porcs, M. Pasteur a cherché un virus atténué de la rage. Les artifices de culture qui avaient servi jusqu'ici pour diminuer l'activité des virus ne pouvaient pas être employés dans les cas de la rage, puisque nous ne savons pas cultiver le virus rabique dans des milieux artificiels, et que nous ne connaissons même pas le microbe rabique. Mais, des générations successives du virus de la rage peuvent être obtenues chez les animaux; si on ensemence, par exemple, le cerveau d'un lapin sain avec une parcelle du bulbe d'un chien enragé, la culture du virus se fait dans l'encéphale et dans la moelle, comme se ferait, dans un bouillon semé avec du sang charbonneux, la culture de la bactéridie du charbon. Ces ensemencements intra-crâniens peuvent être répetés en séries indéfinies et le virus rabique passant ainsi de lapin à lapin s'adapte si bien à vivre dans le système nerveux de cet animal que son évolution devient d'une régularité parfaite. La rage chez un lapin, ainsi inoculé, éclate à heure fixe et la mort survient exactement au moment prévu. Chaque jour on peut avoir ainsi des cerveaux et des moelles épinières qui sont de véritables cultures pures du virus rabique. Lorsque nous cultivons, dans des milieux artificiels, les microbes pathogènes ordinaires, l'aspect de la culture, l'examen au microscope permettent à chaque instant d'en vérifier la pureté; il n'en est plus ainsi avec nos cultures rabiques dans le système nerveux des animaux, nous sommes assurés qu'elles sont pures d'abord par l'évolution régulière de la maladie, ensuite parce que nos cerveaux et nos moelles rabiques ne montrent aucune lésion manifeste, et enfin parce que ni le microscope ni l'ensemencement *in vitro* n'y décélent rien de vivant. N'est ce pas là, Messieurs, une culture originale que celle de ce virus, que l'on ne voit à aucun moment et que l'on peut cependant obtenir en aussi grande quantite que l'on veut et maintenir à un tel état de pureté qu'il est ainsi entretenu, à l'Institut Pasteur, par passages successifs sur le lapin, depuis l'année 1883, sans qu'aucun accident soit venu interrompre cette longue série de générations?

Ce sont les moelles rabiques de passages, dont la virulence est exaltée et fixe, qui fournissent la matière vaccinale pour la prévention de la rage. Il suffit en effet de les disposer à une température de 23°, dans un flacon sterilisé à deux tubulures contenant quelques fragments de potasse et semblable à celui que je vous présente, pour qu'elles se desséchent à l'abri des poussières de l'air et perdent peu-à-peu leur virulence. Après 14 jours elles sont devenues inoffensives; aussi, dans le traitement anti-rabique, on commence par injecter cette moelle de 14 jours, puis on continue par celles de 13 jours et de 12 jours, et ainsi

de suite en allant des virus faibles aux virus forts jusqu'à la moelle de 3 jours qui complète l'immunité. Ces moelles sont broyées dans du bouillon stérilisé, légèrement alcalin, et réduites à l'état de fine émulsion facile à faire pénétrer sous la peau. Les injections sont faites aux flancs alternativement à droite et à gauche.

Quelle est la quantité de moelles rabiques nécessaire pour donner, à un homme mordu, une immunité suffisante sans lui faire courir aucun danger? On conçoit que l'on n'est pas arrivé du premier coup à une formule définitive de traitement. Dans les premier temps de l'emploi de la méthode, on injectait des moelles faibles, c'est-à-dire que l'on commençait par la moelle de 14 jours, pour s'arrêter à celle de 5 jours. Un semblable traitement, efficace dans la plupart des cas, se montrait insuffisant pour les morsures graves ou multipliées. On eut recours alors à des traitements plus intensifs qui ont varié plusieurs fois; mais depuis plusieurs années, la méthode adoptée consiste à donner, les premiers jours, de grandes quantités d'émulsion de moelles faibles, qui sont supportées sans aucun malaise et permettent ensuite d'injecter, sans danger, les moelles actives nécessaires à la production d'un état réfractaire solide. Les doses d'émulsion varient de 3$^{ce}$ pour les moelles faibles à 1$^{ce}$ pour les moelles fortes, la quantité de substance nerveuse sèche employée correspond à peu près à un centigramme pour chaque personne et par injection. Il est évident qu'il ne saurait être question ici d'un dosage absolument rigoureux, puisque, malgré le broyage, les particules en suspension ne sont pas toutes égales. Aussi, on répéte les injections de la même moelle dans le cours du traitement. Celui-ci a donc une certaine durée. Il varie suivant la gravité des morsures.

Pour les morsures ordinaires qui portent sur les membres et ne sont pas trop multipliées, la formule du traitement est la suivante:

| | | |
|---|---|---|
| 1$^{er}$ jour | moelle de | 14 jours à la dose de 3$^{ce}$ d'émulsion. |
| | | 13 " " " |
| 2$^{e}$ | " | 12 " " " |
| | | 11 " " " |
| 3$^{e}$ | " | 10 " " " |
| | | 9 " " " |
| 4$^{e}$ | " | 8 " " " |
| | | 7 " " " |
| 5$^{e}$ | " | 6 jours à la dose de 2$^{ce}$ d'émulsion. |
| | | 6 " " " |
| 6$^{e}$ | " | 5 " " " |
| 7$^{e}$ | " | 5 " " " |
| 8$^{e}$ | " | 4 " " " |
| 9$^{e}$ | " | 3 jours à la dose de 1$^{ce}$ d'émulsion. |
| 10$^{e}$ | " | 5 jours à la dose de 2$^{ce}$. |
| 11$^{e}$ | " | 5 " " |
| 12$^{e}$ | " | 4 " " |
| 13$^{e}$ | " | 4 " " |
| 14$^{e}$ | " | 3 " " |
| 15$^{e}$ | " | 3 " " |

Dans les cas de morsures à la tête, l'incubation de a maladie est parfois très-courte, aussi, faut-il se presser d'agir et multiplier les

injections dans les premiers jours pour gagner du temps. De plus le traitement est prolongé pendant 21 jours, en voici la formule :

| Jour du traitement. | Age des moelles. | Dose injectée. |
|---|---|---|
| 1er jour matin | 14 jours | 3cc. |
| | 13 „ | |
| soir | 12 „ | |
| | 11 „ | |
| 2e „ matin | 10 „ | 3cc. |
| | 9 „ | |
| soir | 8 „ | |
| | 7 „ | |
| 3e „ matin | 6 „ | 2cc. |
| soir | 6 „ | |
| 4e „ - | 5 „ | 2cc. |
| 5e „ - | 5 „ | 2cc. |
| 6e „ - | 4 „ | 2cc. |
| 7e „ - | 3 „ | 1cc. |
| 8e „ - | 4 „ | 2cc. |
| 9e „ - | 3 „ | 1cc, ½. |
| 10e „ - | 5 „ | 2cc. |
| 11e „ - | 5 „ | 2cc. |
| 12e „ - | 4 „ | 2cc. |
| 13e „ - | 4 „ | 2cc. |
| 14e „ - | 3 „ | 2cc. |
| 15e „ - | 3 „ | 2cc. |
| 16e „ - | 5 „ | 2cc. |
| 17e „ - | 4 „ | 2cc. |
| 18e „ - | 3 „ | 2cc. |
| 19e „ - | 5 „ | 2cc. |
| 20e „ - | 4 „ | 2cc. |
| 21e „ - | 3 „ | 2cc. |

Les grandes quantités de moelles faibles, injectées coup sur coup dans les trois premiers jours, ont pour effet d'amener rapidement le patient à supporter les moelles actives que l'on donne ensuite à doses répétées.

Les deux formules que nous venons de faire connaître peuvent être variées suivant les circonstances, c'est ainsi par exemple que dans le cas de morsures profondes et multipliées sur les membres, on applique un traitement intermédiaire entre celui des morsures ordinaires et celui des morsures à la tête.

Les personnes traitées à l'Institut Pasteur sont divisées en trois catégories :—

La première, (Tableau A.) comprend les personnes mordues par des animaux reconnus enragés experimentalement ; c'est-à-dire les animaux dont le bulbe a été inoculé et a donné la rage et ceux qui ont mordu des hommes ou des animaux devenus enragés par la suite.

La deuxième, (Tableau B.) comprend les personnes mordues par des animaux reconnus enragés par des vétérinaires.

La troisième, (Tableau C.) est réservée aux personnes mordues par des animaux suspects de rage.

Le tableau qui est reproduit ici résume les résultats de vaccinations anti-rabiques par catégories et par années. I s'arrête au 1er Janvier

1891, car il ne s'est pas écoulé un temps suffisant pour que nous puissions juger les résultats du commencement de cette année.

| Années. | Tableaux A et B. | | | Tableau C. | | | Total. | | |
|---|---|---|---|---|---|---|---|---|---|
| | Traités. | Morts. | Mortalité. | Traités. | Morts. | Mortalité. | Traités. | Morts. | Mortalite. |
| | | | % | | | % | | | % |
| 1886 | 2,164 | 29 | 1·34 | 518 | 7 | 1·35 | 2,682 | 36 | 1·34 |
| 1887 | 1,518 | 17 | 1·12 | 260 | 4 | 1·54 | 1,778 | 21 | 1·18 |
| 1888 | 1,377 | 11 | 0·80 | 248 | 1 | 0·40 | 1,625 | 12 | 0·74 |
| 1889 | 1,536 | 7 | 0·46 | 298 | 3 | 1·00 | 1,834 | 10 | 0·54 |
| 1890 | 1,330 | 9 | 0·67 | 216 | 2 | 0·92 | 1,546 | 11 | 0·71 |
| Totaux geneeraux | 7,925 | 73 | 0·92 | 1,540 | 17 | 1·1 | 9,465 | 90 | 0·95 |

De 1886 à 1891, 9,465 personnes ont subi le traitement anti-rabique, et parmi elles, 90 ont succombé à la rage, ce qui fait une mortalité de 0·95 %. C'est là, Messieurs, un nombre assurément petit si on le compare à celui de 12 à 14 % qui exprime la mortalité pour les personnes mordues et non traitées. Les chiffres que je viens de citer comprennent tous les morts après traitement; pour être absolument rigoureux, nous devrions retrancher les personnes qui ont succombé dans les jours qui suivent immédiatement le traitement; car chez elles la maladie a éclaté avant que les inoculations préventives aient pu faire leur effet; la mortalité serait alors réduite à 0·61 %. A fin de ne laisser place à aucune critique, nous comptons comme insuccès toutes les morts, quel'que soit le moment où elles sont survenues.

Pour mieux juger de l'efficacité du traitement anti-rabique, négligeons toutes les personnes blessées par des animaux suspects de rage, pour ne considérer que celles qui ont été mordues par des chiens reconnus enragés, et qui figurent dans les Tableaux A et B. Bien plus, choisissons dans cette catégorie celles qui ont été mordues à la tête. Vous lavez tous, Messieurs, qu'elle est la gravité des morsures faites à la tête par des chiens enragés. Elles entraînent chez les personnes non traitées une mortalité de 80 %. Parmi nos traités, nous trouvons 710 personnes qui ont subi de semblables morsures, elles ont donné 24 morts, c'est-à-dire que leur mortalité a été seulement de 3·38 %. L'écart entre ces deux chiffres 80 % et 3·38 % mesure la valeur des inoculations préventives. Et encore dans ce nombre de 3·38 % sont compris les cas où la rage a éclaté aussitôt après les inoculations préventives et qui légitimement pourraient être déduits.*

Est-il possible, Messieurs, de donner une preuve plus convaincante de l'efficacité de ce traitement anti-rabique que l'on a cependant dénoncé comme insuffisant et dangereux? En présence de semblables

* Neuf personnes ont succombé à la rage dans les 15 jours qui ont suivi le traitement. Si on les retranche nous avons seulement 15 morts sur 700 traités, soit une mortalité de 2·14 %.

résultats, que pèsent, je vous le demande, les objections qui ont été faites à la méthode Pastorienne? Quelle est, parmi les médications les plus sures de la médecine, celle qui ne compte pas plus d'insuccès? Nous n'avons donc qu'une chose à répondre aux détracteurs de parti-pris :—avez-vous quelque chose de mieux à proposer?

Parmi les 9,465 personnes qui sont venues réclamer le traitement contre la rage il y en a 1551 étrangères à la France et appartenant à vingt nationalités diverses. Elles ont donné 24 décès par rage, soit 1·29 °/₀. Cette mortalité, plus élevée que la mortalité générale, s'explique par l'éloignement des personnes mordues qui souvent ne peuvent être soumises au traitement que long temps après la morsure.

Il vous paraîtra peut-être interessant de savoir que 389 personnes de nationalité anglaise out été traitées à l'Institut de Paris, la mortalité s'est élevée sur elles, à 1·8 °/₀; car beaucoup qui avaient subi des morsures très graves ne se sont présentées que plusieurs jours après leur accident. Il ne faut pas oublier, en effet, que le traitement réussit d'autant mieux qu'il est entrepris plutôt après les morsures.

Qu'est devenu le virus rabique dans les moelles desséchées? Pour répondre à cette question, inoculons à des lapins, par trépanation, les moelles d'âges divers qui servent aux injections préventives. Nous voyons que déjà après cinq jours elles sont le plus souvent inoffensives. C'est l'action de la dessiccation et de l'air qui a modifié ainsi le virus rabique, en effet les moelles, maintenues à la même température de 23°, dans une atmosphère humide d'acide carbonique ou d'hydrogène, restent longtemps virulentes. Cette modification est-elle une atténuation véritable analogue à celle de la bactèridie du charbon transformée en vaccin charbonneux, et capable de se maintenir avec ses caractères? Il ne paraît pas en être ainsi, car lorsqu'on arrive à faire périr lapin de la rage en lui injectant de très grandes quantités d'une moelle faible, le virus que l'on trouve dans son cerveau et dans sa moelle manifeste d'emblée la virulence maximum du virus de passage. On ne peut donc pas dire que chacune de nos moelles représente, suivant son âge, un dégré particulier d'atténuation. Il est probable que dans moelles rabiques le virus est d'abord modifié dans sa vitalité, puis qu'il périt en grande partie sous l'influence de l'air et de la dessiccation, sans avoir subi une atténuation véritable. Mais comme l'action de la sécheresse et celle de l'air n'exercent pas également dans toute l'épaisseur de la moelle le virus n'est pas également réparti dans toutes les portions. Selon la dimension des moelles, les modifications seront plus ou moins rapides. Aussi, dans la pratique emploie-t-on des lapins presque toujours de même poids (2 kilog.) qui donnent des moelles sensiblement égales. C'est à cause de cette inégale répartition du virus qu'il faut répéter les injections et donner de fortes doses des moelles les plus âgées. Il semble que la vaccination anti-rabique soit ainsi plus délicate à appliquer que la vaccination charbonneuse, par exemple, qui se fait au moyen de microbes amenés à un degré de virulence précise, qui peut se pérpétuer dans les cultures. Cependant, si on se conforme aux indications que nous avons données, la pratique des inoculations préventives contre la rage est très facile à conduire.

A côté du virus vivant, mais modifié, que renferment les moelles desséchées, M. Pasteur pense qu'il existe des produits chimiques spéciaux élaborés par le microbe de la rage et qui entrent pour une part dans la production de l'immunité, de sorte que dans les inoculations anti-rabiques il y aurait à la fois vaccination chimique et vaccination par virus-vivant. De nombreuses expériences sont encore nécessaires pour élucider cette question ; mais, ce qui est acquis, par des essais multipliés sur les animaux, et par la pratique sur l'homme, c'est qu'il est nécessaire pour donner une immunité solide et durable, de terminer par des inoculations de moelles actives. Des chiens qui avaient reçu jusqu'à la moelle de o jour avaient encore l'immunité après plus de trois anneés. Chez l'homme nous ne dépassons pas la moelle de 3 jours dont l'activité est suffisante, surtout si elle est injectée plusieurs fois ; cependant dans les cas tout-à-fait exceptionnels on peut donner pour plus de sureté, la moelle de deux jours.

Les personnes qui suivent le traitement contre la rage, ne ressentent pour la plupart aucun malaise. Quelques unes se plaignent de fatigue, d'énervement et parfois de somnolence, mais nous n'avons jamais relevé chez elles de symptômes plus ou moins analogues à ceux de la rage, et qui puissent être rapportés aux injections qu'elles ont subies. D'ailleurs, beaucoup de médecins, non mordus par des chiens enragés, ont pratiqué sur eux, par manière d'expérience, les inoculations anti-rabiques, la grande majorité n'en a éprouvé aucun effet ; d'autres ont ressenti de l'énervement et une lassitude marquée pendant plusieurs jours.

Il arrive parfois que des personnes gravement mordues, surtout celles qui sont venues à l'Institut un temps assez long après les morsures, présentent des symptômes qui font craindre l'apparition de la rage. M. Laveran et M. Chautemesse ont cité des observations où les patients se plaignaient de douleurs sur le trajet des nerfs, partant des blessures, de faiblesse et d'hypéresthésie du membre mordu, et montraient de même de l'aérophobie et de la difficulté à déglutir. Puis, tous ces signes disparaissaient, comme s'il s'agissait d'une rage avortée sous l'influence du traitement. De semblables observations sont plus difficiles à faire sur les animaux, cependant nous avons vu des chiens traités après inoculation qui à un moment donné, devenaient furieux et avaient la voix rabique, ces symptômes persistaient pendant quelques heures où même quelques jours et cessaient bientôt. M. Högyes a cité des faits analogues observés sur des chiens, dans le cours de ses expériences. Il semble donc que la rage, après avoir commencé, puisse guérir dans certains cas.

Un point, sur lequel il convient d'insister, c'est que dans les diverses manipulations qu'elles subissent, les moelles qui servent aux injections doivent rester absolument pures. La présence de microbes étrangers causerait des suppurations chez les personnes traitées qui reçoivent des inoculations répétées, et pourrait de plus entraver l'action du virus préventif. La pureté des moelles est vérifiée par leur ensemencement en bouillon, d'abord au moment où elles sont introduites dans le flacon desséchant, et ensuite deux jours avant leur emploi. Chaque moelle est ainsi accompagnée de deux flacons de bouillon qui

doivent rester parfaitement limpides et qui sont comme les témoins de sa pureté. En opérant ainsi, on peut éviter presque complètement les abcés ; malgré le nombre immense d'inoculations faites à l'Institut Pasteur, les suppurations sont très rares, et si elles se montrent on peut être assuré qu'une négligence a été commise.

Depuis l'origine du traitement anti-rabique, la mortalité a diminué, parce que nous avons appris à mieux nous servir de la méthode ; cette mortalité qui en 1886 atteignait 1·34 °/₀, est tombée à 0·54 °/₀ en 1889, et n'a pas dépassé 0·71 °/₀ en 1890.* Peut-on espérer, Messieurs, que cette mortalité deviendra nulle ? Je ne le crois pas. Parmi les personnes traitées qui ont succombé, beaucoup ont pris la rage dans la quinzaine qui a suivi les inoculations ; c'est-à-dire que chez elles, le virus rabique a atteint les centres nerveux, peu de temps après la morsure, pendant le traitement. Dans ces conditions, les injections préventives, faites loin de l'axe cérébro-spinal, n'ont pu modifier à temps la moelle et l'encéphale, et empêcher la culture du virus rabique. Une autre cause d'insuccès, c'est la longue dureé du virus rabique dans le corps ; il peut, en effet, s'y conserver à l'état latent pendant des mois, et même des années, puis, à la suite d'une cause banale, la rage éclate. L'état réfractaire produit par les inoculations préventives peut cesser avant que le virus ait péri, on comprend alors que même après le traitement il puisse avoir des rages tardives. Ces cas sont heureusement fort rares, et la méthode anti-rabique donne la sécurité contre la rage dans l'immense majorité des cas.

Messieurs, que de difficultés vaincues dans cette étude de la rage ? La vaccination anti-rabique est un des exemples les plus éclatants de ce que peut la méthode expérimentale appliquée aux choses de la médecine. Cette période d'incubation de la rage, qui était pour les mordus un temps d'angoisses, est mise à profit pour assurer leur immunité ; ce virus resté inconnu est préparé à l'état de pureté et transformé en vaccin. Ce qui caractérise les découvertes de M. Pasteur, c'est qu'à peine sorties du laboratoire elles entrent dans la pratique. La méthode de prévention de la rage n'est pas seulement appliquée en France, des instituts, sur le modèle de celui de Paris, existent en Russie, en Italie, en Turquie, au Brésil, aux Etats-Unis, etc. Combien, sur les milliers de personnes qui ont été traitées, doivent la vie à la vaccination Pastorienne ?

Pour atteindre à un semblable résultat il a fallu, pendant plusieurs années, les efforts réunis de M. Pasteur et de ses collaborateurs, et cependant il serait si facile de faire disparaître la rage. Il suffirait que les pays voisins soient d'accord pour appliquer les règlements sanitaires avec autorité et persévérance pendant quelques mois. Mais, il y a une passion plus forte que les règlements sanitaires et souvent que la raison : c'est la passion des chiens. Le propriétaire d'un chien n'accepte pas que son chien puisse devenir enragé, il se refuse à l'abattre et même à le museler, quand il a été mordu. Il admet bien que le chien du

* Voir les divers rapports publiés par M. Perdrix dans les Annales de l'Institut Pasteur.

voisin est capable de tout, voire de la rage; le sien ne doit pas être soupçonné. Ce qu'il faudrait, c'est un changement dans les mœurs, chacun de nous doit s'y employer.

Soyez assurés que nous rencontrerons dans cette tâche des difficultés plus grandes que celles surmontés par M. Pasteur dans l'étude de la rage. C'est pourquoi, Messieurs, le traitement anti-rabique sauvera encore bien des existences, avant que les progrès de l'hygiène l'aient rendu inutile.

## The Propagation and Prevention of Rabies.

BY

GEORGE FLEMING, C.B., LL.D., F.R.C.V.S.

It is now generally accepted by those who have most carefully studied the subject, that rabies is a purely contagious disease, due to a virulent principle or specific agent, and that it is, as a rule, transmitted from a diseased to a healthy creature by inoculation. The specific agent, when introduced into the body of a susceptible animal, more particularly localises itself in the nervous system, and after a certain period, which is somewhat indefinite in accidental inoculation but definite in experimental inoculation, causes such disturbance in the functions of the brain and spinal cord as to give rise to well marked symptoms and to a morbid condition which, it may be asserted, invariably terminates in death.

Inoculation and infection occur through wounds, which are generally inflicted by the teeth of creatures affected with rabies.

Of all the domesticated animals which may become affected, the dog is certainly the one that is by far the most frequently attacked, and it is the one which perpetuates the malady; it is undoubtedly the chief, if not the only propagator of the scourge in this and other countries; and it is, therefore, exceedingly probable, nay, certain, that if the canine species were completely freed from rabies, the disease would be no longer known.

Recognising this fact, and in view of the constant virulency of the saliva of rabid animals, and consequently of danger from their bites, and the serious consequences resulting therefrom, the importance of sanitary measures applicable to this animal cannot be over-estimated. In the interests of mankind, no less than in those of the domesticated animals, and particularly of the dog itself, this very alarming, most painful, and always fatal disorder should be suppressed. And of all the known contagious diseases, rabies is the one which can be most easily and quickly extinguished, provided proper measures are prescribed and are carried stringently and energetically into operation.

The measures necessary for the extinction of the disease must be based on the fact that the dog is the chief, nay, sole, propagator of rabies; that the contagium or infecting material is conveyed from the

diseased to the healthy by means of bites (other means are so exceedingly rare that they may be left out of consideration); and that to free a country from it, all rabid and suspected dogs should be destroyed, certain restrictions maintained for a limited period, and only dogs from other countries exempt from rabies allowed admission. Dogs coming from infected or suspected countries, should undergo a period of quarantine equal at least to the longest period of latency of the disease.

That the disease can be limited, or altogether suppressed by the enforcement of proper measures, there is an abundance of evidence to prove. Sweden, Norway, Switzerland, Baden, Prussia, Bavaria, Wurtemburg, and other countries have been freed from it by such measures; while countries in which it has never been seen, as Australia and New Zealand, are preserved from its invasion by such precautionary measures as quarantine of imported dogs, or, as in Sweden, total prohibition of importation of dogs.

That the dog is the chief sufferer from, and, it may be asserted, the sole propagator of the malady, is amply proved by every-day experience; but the following table, showing the number of cases of rabies in England during the four years 1887–90, and the number and species of animals affected, affords further evidence:—

| Kind of Animal. | 1887. | 1888. | 1889. | 1890. |
|---|---|---|---|---|
| Dogs | 217 | 160 | 312 | 129 |
| Cattle | 11 | 2 | 9 | 2 |
| Sheep | 5 | 7 | 11 | — |
| Swine | 3 | — | 4 | 1 |
| Horses | 4 | 5 | 4 | 2 |
| Deer | 257 | 2 | — | — |
| Total | 497 | 176 | 340 | 134 |

It will be observed that in 1888 and 1889, 90 and 91 per cent. of the animals reported as rabid were dogs, and in 1890 the per-centage was more than 96. The animals other than dogs were infected by rabid dogs, and these infected each other; as the mad dog has always the strongest tendency to attack its own species, it follows that this furnishes by far the largest proportion of victims.

The suppressive sanitary police measures are, in the order of their importance:—1. Destruction of all dogs which are rabid, or which are suspected of being or becoming rabid; 2. The seizure and, if need be, destruction of all ownerless and wandering dogs; 3. All other dogs to wear a properly constructed and well-fitting muzzle while rabies prevails, and also for a period equal to the longest interval of latency after the malady has been suppressed; 4. The imposition of a tax upon all dogs.

1. The necessity for the destruction of all rabid dogs is so self-evident that it need not be further insisted upon. The disease is incurable, causes much suffering, and the affected dog is, so long as it

lives, so great a danger to other creatures that its speedy destruction is imperative. Dogs which are suspected of being rabid, or of becoming so, through contact with those which have been affected, must always be a source of grave apprehension and danger, and unless they can be kept under close and safe observation for a considerable time, should also be destroyed, seeing the risks incurred in allowing them to live.

A suspected dog may be defined to be one that presents symptoms resembling those of rabies, or which has been in conditions that have rendered infection probable or possible.

2. Wandering dogs are the chief agents in the propagation of rabies, for the reason that they are more exposed to contamination than those which are properly cared for, and also because rabid dogs, even when carefully guarded, seek to wander from home. In proportion as wandering dogs abound in infected countries, so is the disease prevalent. This is a well-established fact. In England in 1888, of 160 rabid dogs, 64, or 40 per cent., were returned as stray ones; in 1889, of the 312 rabid dogs reported, 121, or more than 38 per cent., were evidently ownerless; while in 1890, 64 out of 129, or more than 51 per cent., were so returned.

Therefore, in order to limit the propagation of rabies, all wandering dogs should be secured, and, if not claimed within a certain period, destroyed.

3. Rabies being transmitted, as a rule, by the bites of dogs, and as in countries in which the disease prevails there can be no certainty when a dog is not infected, if it at all times be prevented from inflicting wounds by its teeth, it is evident that the danger of extension of the malady must be reduced at least to a minimum. Dogs can be hindered from biting by causing them to wear a properly constructed muzzle; and if it fits well, it should cause very little, if any, inconvenience to them.

Rabid dogs sometimes escape from home without their muzzles, and the absence of these is an indication that they should be seized; and wandering and ownerless dogs are also known by their not wearing muzzles, and can therefore be secured by the police.

The value of the muzzle in suppressing rabies has been demonstrated on many occasions; and in serious outbreaks of the disease, its introduction has always coincided with a diminution in the number of cases, and the eventual extinction of the scourge. Its use is also coincident with a decrease in the number of cases of hydrophobia occurring among people during such outbreaks, and their ultimate cessation.

The evidence in support of this statement is so voluminous and explicit, that anyone examining it must be convinced as to the certainty of the fact. Two or three instances may be cited.

In the Report of the Royal Commission on Rabies, it is stated that "in the city of Berlin special regulations are in force. In con-
" sequence of a severe outbreak in the year 1852, during which 107

" dogs were destroyed as rabid, the Royal Police issued a decree to " the effect, on July 2, 1853, that all dogs should be provided with a " wire muzzle positively preventing the animal from biting, and to " empower special persons appointed by the police for that purpose " to seize and destroy all dogs not so muzzled; and, when the owner " could be found, imposing a fine of ten thalers (1*l.* 10*s.*) or a term " of imprisonment. In the year following this decree only one dog " was killed as rabid, against 97 in the previous year. The decree " still remains in force, but does not seem to have been effectual in " preventing the recurrence of epidemics of rabies; for the number " of dogs killed as rabid, which up to 1863 had not exceeded nine " in any year, rose progressively in the succeeding years, till in 1868 the " number had reached 66, declining again to seven in 1870, only to " increase in 1872 to 69. In 1875 a law was passed, extending to the " whole of Prussia, for the suppression and prevention of animal " disease, which provides that all dogs suspected of rabies shall be " immediately killed, as also all animals which it is evident have been " bitten by rabid animals; and that all dogs in a district which has " been infected by an outbreak of rabies shall be confined, or, when " abroad, both muzzled and led. The technical section of the Veterinary " Board in Berlin are of opinion that the passing of this law, and not " only the existence of the muzzling order in that city, is the cause " of the extinction of rabies in Berlin; no case has occurred there " since 1883."

In Vienna, we are informed that rabies was entirely suppressed by 18 months of stringent muzzling; but in the summer of 1886 the muzzling order was rescinded, and badges had to be worn on dogs' collars instead. In the following half-year there was only one case of the disease, but in the next half-year rabies became epizootic and the muzzle had again to be worn, with the result that the malady soon subsided and disappeared. Consequently, the muzzling order is still rigidly and most beneficially enforced in Vienna.

In Holland, before 1875, rabies was prevalent to a very serious extent; but in June of that year the use of the muzzle was ordered, with the result that in the autumn the number of cases fell to 41; in the next whole year they were 55; in 1877, they were 14; in 1878, they were four; and in 1879, they were three. These and the cases which have since been reported, only occurred on or near the frontier of Belgium, in which country the muzzle is not in use, though rabies is always prevalent. To such a degree, indeed, does the disease exist in Belgium, that in 1889 there were brought to the Veterinary School at Brussels no fewer than 94 really rabid dogs, 49 having been admitted during the winter quarter, and 45 during the summer quarter; and from January 1st to April 24th, 1890, 16 were taken there.

In the Grand Duchy of Baden, during the years 1871, 1872, 1873, 1874, and 1875, the number of cases of rabies were respectively 18, 37, 37, 50, and 43. Then the muzzle was rigorously applied, and in 1876 there were 28 cases; in 1877, three; in 1878, four; in 1879, two;

in 1880, two; in 1881, two; in 1882, three; in 1883, two; in 1884, two. Since that year only one case has been observed, and that was a dog from Metz, where there were no proper regulations, which had been contaminated before its arrival at Baden.

In Sweden, rabies was at one time a somewhat common disease, and from eight to ten people died annually of hydrophobia; but—muzzling being enforced and the importation of dogs prevented—rabies has been unknown for many years, and no deaths from hydrophobia have occurred since 1870.

In England, the value of the muzzle has been as well exemplified as elsewhere, though its application has always been extremely partial, being only employed in towns or districts when rabies has increased to such an extent as to create alarm, and removed immediately when the disease had for the time disappeared. No effective action has at any time been taken to suppress rabies in England or Ireland; in the latter country it is always more or less prevalent, and ever since it was first legislated for in England it has maintained a permanent existence in the following 14 counties, viz., Chester, Derby, Essex, Hants, Kent, Lancaster, London, Middlesex, Notts, Surrey, Sussex (East), Sussex (West), Warwick, and York (W.R.). During the year 1890–91 cases of rabies were reported for the first time in three counties previously free from it; these were Bucks, Norfolk, and York (E.R.); and it was re-introduced into Leicester after an absence of two years, and into Stafford, which had been free for one year.

The extent to which rabies has prevailed in this country may be judged from the loss of human life through bites from rabid dogs. It is stated that in England (including Wales), there have been 939 deaths from hydrophobia recorded during the past 38 years, the yearly average for the first 16 years being eight, for the next 16 years 15, and for the remaining period ending in 1885, 45. Thus, the mortality has steadily advanced through more than 400 per cent. On the other hand, the Prussian preventive measures have reduced deaths from hydrophobia to a remarkable degree; for while, in the decade ending in 1819 there was a yearly average of 166 deaths; in a similar period ending in 1886, there was a yearly average of $4\frac{1}{2}$.

The value of the muzzle in suppressing rabies has been, perhaps, best demonstrated in London on several occasions, and especially in 1885. In the previous years, hydrophobia had increased to a very alarming extent, as has just been mentioned, in England, and no steps worthy of note had been taken to check the mortality. For London alone in that year, no fewer than 27 deaths of people were reported as due to the bites of rabid dogs. A muzzling order was then enforced, and at the end of 1886 not a death was recorded. Unfortunately, the order prescribing the use of the muzzle was then rescinded, and in a few months a case of hydrophobia occurred in the south of London, soon to be followed by others, and in 1889 ten deaths were registered. In July of that year the muzzling order was again issued and stringently carried out, and rabies and hydrophobia once more disappeared.

In other countries where rabies prevails and dogs are not muzzled, though other measures, as the dog-tax, medal on the collar, leading by a leash, &c., are enacted, the malady continuously manifests itself, and numbers of people perish from hydrophobia every year. We may give Belgium and France as examples. In the latter country the monthly sanitary bulletin shows to what an extent it is prevalent, and I need only refer to the two last which I have to hand, those for March and April of the year 1891. In March, 132 dogs and eight cats were destroyed as rabid, besides those sacrificed as a preventive measure; while 32 persons, as well as cattle and pigs, were bitten by mad dogs. In April, 151 dogs and four cats were destroyed because of being rabid, and a large number were killed because they had been bitten by mad dogs, or were wanderers; and 47 persons were wounded by mad dogs. The reports of the Pasteur Institute show that by far the largest proportion of persons protectively inoculated are French. There can be no doubt that if the use of the muzzle were enforced generally and strictly throughout France, rabies would quickly vanish from the sanitary bulletins.

Belgium has tried all the other recognised measures except the muzzle, and yet the malady is as rife and deadly as ever in that country. A Royal Commission was recently appointed to inquire into the subject, and the report addressed to the Superior Council of Hygiene in April last states that the regulations in force are insufficient, and while not contesting the value of Pasteur's preventive inoculations with regard to people bitten by rabid dogs, it is urged that there is something far more desirable, and that is the extinction of the disease in the canine species. For this object, the Commission insists upon the immediate adoption of the muzzle.

Senseless sentimentality has opposed the use of this article in a most extraordinary manner in this country, and one would be inclined to believe that there are people who care less for human suffering and human life than for a little inconvenience or discomfort to dogs. A well-fitting muzzle should cause very trifling inconvenience and discomfort, while ensuring absolute safety from dog-bite. Repeatedly, rabid dogs have been brought to veterinary surgeons, wearing muzzles, and so rendered safe; as it is a well-known fact that a diseased dog will, in nearly every instance, allow those it knows to handle it and put on a muzzle. A leash only is no protection, for the dog can bite and is as dangerous as if not led. Frequently rabid dogs on the leash are brought to veterinary surgeons. It is also perfectly obvious that a collar, no matter how embellished and be-medalled it may be, will no more prevent rabies, or hinder dogs from biting, than will a linen collar on a man's neck preserve him from small-pox or influenza. But we need not stay to notice these ridiculous notions.

4 The imposition of a dog-tax and registration are necessary to limit the number of dogs, and to ensure that every one has an owner, who should be held responsible for any damage it inflicts. From this measure no dog-owner should be exempt.

If an entire country is infected with rabies, then suppressive measures should be applied to the whole area, uniformly and energetically, until the malady is perfectly extinct. If only a portion of a country is visited by it, then it is questionable whether such measures should be limited to that portion only. If it were possible to keep all the dogs it contains within its boundaries, then such a step would suffice, if a certain zone around it were established beyond which no infected animals should pass. But it would be extremely difficult, if not impossible, to establish such a barrier; and to effectively rid a country of rabies, though only partially infected, the sanitary police measures should be applied to the whole.

That such a result is possible has been proved by the experience of the countries I have named; and from these examples, and from what we now know of the disease, it may certainly be affirmed that rabies need only exist in a country which does not desire to get rid of it. The United Kingdom can quickly and easily free itself from it, and keep itself free if it cares to do so; and a heavy responsibility for the loss of human life rests upon those who oppose, or do not choose to adopt, the measures indicated. Continental nations with co-terminous frontiers should combine in a simultaneous effort to abolish a scourge which creates so much dread, and causes so much suffering and terrible death to man and beast. Such a consummation can be realised; and it only needs the will to effect it.

## Études sur la Rage et sur la Vaccination antirabique.

PAR

le Professeur V. Babes, Bucarest.

[This Paper was originally contributed to Section II.]

Les recherches sur la vaccination antirabique ont été pour ainsi dire, terminées quand M. Pasteur eut commercé le traitement antirabique chez l'homme; il ne restait presque que des questions d'un intérêt secondaire, mais qui cependant avaient leur importance dans la pathologie de la rage, dont l'étude est devenue facile, grâce aux découvertes épocales de M. Pasteur.

*Etiologie et histologie.*—Nous avons d'abord étudié la rage au point de vue de son étiologie, mais jusqu'à présent nos efforts sont restés sans résultat, le microbe de la rage n'est pas trouvé, et il faut se demander si avec nos moyens actuels de recherches nous le trouverons. Les méthodes connues de culture ne donnent pas un résultat concluant, et le microscope ne nous montre pas des parasites analogues aux microbes. Il est vrai que dans les préparations fraîches ou colorées avec toutes les méthodes perfectionnées, on trouve beaucoup d'éléments suspects, même plus ou moins caractéristiques, mais pas un seul de ces éléments ne nous donne des garanties pour son rôle essentiel dans cette maladie.

Il s'agit en partie de formations ressemblant aux corpuscules hyalins ou protoplasmatiques, comme celles, par exemple, qu'on avait regardées comme les parasites du carcinome ou des différentes maladies de la peau.

Quoique une telle interprétation n'est pas justifiée dans la rage, ces corpuscles, en même temps que certaines lésions particulières du système nerveux central, forment un ensemble assez caractéristiques, de sorte qu'on peut en profiter pour le diagnostique plus rapide de la maladie du chien mordeur. Ces lésions et formations sont les suivantes :—

Les lésions inflammatoires qui ont été déjà décrites en partie par Benedikt, Kolesnikoff, Coats, et Gombault. Mes recherches publiées en 1886, ont montré que les lésions sont plus ou moins localisées dans les cornes antérieures de la moëlle, à la base de la fosse rhomboïdale, surtout dans les noyaux du hypoglosse et le long du raphée, de même que, dans certains centres moteurs du cerveau. Ces lésions, dont quelque unes ont été encore décrites plus tard par MM. Schaffer et Popoff, sont les suivants :—

(*a.*) Hypérémie, diapédèse des globules blancs et rouges avec prolifération des cellules périvasculaires, avec une exudation particulière séreuse avec dilatation des espaces lymphatiques.

(*b.*) Petites hémorrhagies à la surface du plancher du IV$^{ème}$ ventricule dans le voisinage du canal central de la moëlle et dans les cornes antérieures. Les vaisseaux des foyers hémorrhagiques sont souvent oblitérés par des masses de leucocytes et des cellules resemblant aux leucocytes, mais avec certaines particularités (petits noyaux ? fusiformes, avec des parties pâles au milieu, pigmentation, etc.), ou bien remplis de masses hyalines.

(*c.*) On trouve dans la rage des granulations chromatiques particulières dans l'intérieur des leucocytes libres ou dans l'intérieur des vaisseaux, autour des vaisseaux, mais surtout dans les hémorrhagies, le long de certaines fibres motrices et dans le canal central.

(*d.*) Dans les mêmes endroits il existe encore des globules et des masses hyalines de différentes grandeurs.

(*e.*) On trouve toujours autour de certains vaisseaux et dans certains groupes de cellules nerveuses du bulbe et des cornes antérieures de la moëlle, des régions indiquées des noyaux miliaires embryonnaires avec dilatation des espaces lymphatiques et avec un réseau particulier chromatique.

(*f.*) La lésion des cellules motrices présente certaines particularités. Elle est la plus prononcée au milieu de ces nodules milliaires, et elle consiste d'abord dans un dérangement du réseau chromatique mis en évidence par le bleu de Löffler, dans le protoplasme de la cellule, d'une infiltration de pigment, ou des masses granuleuses, Karyo-kinèse ou fragmentation du noyau. Les lésions plus prononcées consistent dans l'apparition des grands espaces sinneux péricellulaires, infiltration de leucocytes, non seulement dans ces espaces,

mais aussi dans la cellule même, disparition du réseau chromatique et du noyau, uniformisation avec rupture des prolongements et atrophie.

(*g.*) La lésion des tubes nerveux est moins prononcée. J'avais constaté un œdème de la gaine myélinique, surtout de certains tubes moteurs et dans le liquide qui se trouve entre la myéline et le cylindre axe nagent des corpuscles ronds, souvent doubles, mal colorés par les couleurs d'aniline et par l'acide osmique et qui semblent posséder un mouvement propre assez énergique. Dans des préparations absolument fraîches ou reconnaît au plancher, des plus grandes cellules possédant une motilité énergique.

(*h.*) Au contraire nous ne pouvons pas confirmer ni la spécificité de certaines granulations ou lésions décrites par Schaffer, ni la localisation exacte des lésions à l'endroit de l'entrée du nerf de la région mordue, localisation prétendue de cet auteur.

Par des méthodes de durcissement et de coloration expéditives, par exemple, liqueur de Flemming, tannin, et fuchsine de Löffler, on peut avoir des préparations démonstratives, trois jours après la mort du chien mordeur et les lésions décrites nous serviront en même temps que les symptômes et l'autopsie du chien pour le diagnostique de la maladie.

Comme le résultat de toutes nos recherches sur le virus, de la rage, malgré de centaines d'essais de culture, a été toujours négatif, nous nous sommes demandés si le virus, de même que dans le tétanus ne reste pas dans la plaie sans entrer dans l'économie. Cette supposition serait en contradiction avec le fait que le système nerveux n'est pas virulent dans le tétanus, tandis qu'il l'est toujours dans la rage.

Je connais cependant quelques faits où les organes internes des tétaniques ont possédé une certaine toxicité, et où cette toxicité a pu être transmise à plusieures générations. Il est vrai que des recherches de contrôle de ce fait curieux ont été toujours négatives. Toutefois, si on trouvait des substances chimiques produites par une bactérie, mais indépendantes jusqu'à un certain degré, et qui sans la concurrence continue du microbe pourrait transformer une matière renfermée dans le système nerveux en toxine spécifique, le problème de l'étiologie de la rage, et, peut-être, aussi d'autres maladies infectieuses, serait résolu. Il est certain que les faits bien connus concernant l'étiologie des maladies infectieuses, ne donnent pas d'appuis à cette idée.

*Substances chimiques produites par le virus rabique.*—Le fait principal qui s'oppose à une telle supposition est l'impossibilité de tirer des organes rabiques une substance qui puisse produire les symptômes de la rage.

Toutes les tentatives faites dans cette direction par le chimiste de notre institut, M. A. Babes, et de moi même-ont été négatives, et il en résultait seulement :—

(1.) Qu'il existe une substance toxiques dans le système nerveux de l'homme et des animaux morts de la rage. (2.) Que cette substance obtenue

par la filtration sous pression et par précipitation dans l'alcol, ou mieux encore par dialyse, est soluble dans l'eau et glycérine; elle présente d'analogies avec les enzymes et paraît être complexe. (3.) Cette substance possède à l'état frais une action très violente et produit chez le chien, le lapin, le cobaye, même en très petites doses de la fièvre, une hypéresthésie, des paralysies et la mort, mais sans les symptômes pathognomiques de la rage. Son action est seulement générale. (4.) Les lapins et les chiens peuvent s'habituer à l'effet des doses croissantes de ces substances et les lapins gagnent par ce traitement une résistance un peu plus grande à l'infection rabique, mais aucun animal n'avait pas gagné par ce procédé l'immunité contre la rage.

*Destruction du virus rabique.*—Une question de grande importance nous préoccupait encore avant d'entrer dans l'étude de la vaccination antirabique, à savoir, si on ne pouvait pas par la destruction du virus rabique dans la plaie, empêcher la résorption du virus. Tout d'abord nous avons essayé de tuer le virus rabique par des désinfectants et par la chaleur, et nos recherches, publiées en 1886, nous ont montré que la moëlle rabique possède une résistance particulière à l'action de ces agents, ce qui tient principalement au milieu gras dans lequel se trouve le virus. Il faut l'action durant plusieurs heures d'une solution de sublimé 1·1,000 ou de 1·100 d'acide phénique pour rendre le virus rabique inoffensif. Les accides forts et même l'alcol ont une action plus énergique; la température de 58° tue le virus dans une heure, la température de 60° C. en quatre minutes. La lymphe des grenouilles, ou le sang des chiens immuns, mêlés avec une émulsion filtrée par le papier Joseph, tue le virus fixe en 5–6 heures, après l'avoir graduellement atténué. Pour empêcher la manifestation des symptômes rabiques après l'infection il a fallu amputer la partie infectée à peu près un quart d'heure après l'infection. L'amputation du nerf de cette région, même après l'infection dans le nerf même, n'empêche pas le développement de la maladie. En cauterisant la plaie avec le fer rouge, cinq minutes après la morsure, on peut arrêter l'action du virus et même en cauterisant 10 jusqu'à 15 minutes après la morsure, on retarde la manifestation de la maladie. Les caustiques et les substances antiseptiques (acides forts, acide phénique pur ou dilué) n'empêchent pas le progrês de la maladie si on les applique sur la plaie plus tard que cinq minutes après l'infection. Le sang des animaux immunisés et de la grenouille inoculé en même temps, ou mêlé avec le virus rabique, n'empêche en rien l'infection. De même l'inoculation d'autres microbes: streptocoques, prodigiosus, bacilles du pus bleu, ou bien leurs produits solubles, en même temps que le virus rabique, avant ou après cette infection, n'empêchent non plus la manifestion de la rage. Ces recherches nous indiquent de nouveau que c'est ce virus même qui doit nous fournir des résultats pratiques.

*Propagation du virus rabique.*—En ce qui concerne la marche de l'infection il faut que j'insiste sur la priorité de mes recherches sur l'infection dans les nerfs et sur la propagation du virus rabique par les nerfs, dont la preuve principale était non seulement la sureté et la

rapidité de la manifestation de la maladie, en inoculant dans un nerf où dans une plaie intéressant un grand nerf, mais la virulence précoce du nerf infecté, tandis que ordinairement les nerfs éloignés du centre ne sont qu' exceptionellement virulentes. Pendant l'inoculation de la rage j'ai découvert chez le lapin des manifestations fébriles intermittentes ou remittentes qui nous indiquent le travail du virus rabique. J'ai essayé ensuite de profiter de cette fièvre pour constater si après la morsure chez l'homme le virus rabique est en effet entré sons une forme efficace dans l'économie. Ces travaux sont presque impossibles dans des circonstances ordinaires, mais en 1888, ils se sont présentés des cas qui se prêtaient à ces recherches. C'était au commencement de nos inoculations antirabiques chez l'homme que se sont présentés 14 hommes mordus d'une manière terrible à la face par un loup enragé. Notre statistique antérieure au traitement Pasteurien montrait dans de tels cas une mortalité de 90 °/₀. Ces individus sont venus six jours après la morsure, et j'ai commencé immédiatement un traitement intensif d'après les indications spéciales de M. Pasteur. En même temps j'ai pris deux fois par jour la température de ces individus. Un de ces hommes, mort à la suite de ses plaies terribles, deux jours après la morsure n'entre pas dans le cadre de ces recherches. Parmi ces mordus six sont morts de la rage et sept ont été sauvés. Les personnes sauvaient avaient une faible élévation de température 0, 1, 1, 2, 2, 3, 3 fois pendant le traitement, tandis que ceux qui ont succombé montraient une élévation de température, 5, 5, 5, 5, 6, 7 fois pendant les 20 jours qui avaient suivi la morsure. Il faut cependant remarquer que ces individus n'ont pas eu la fièvre qu'on remarque chez les lapins inoculés avec le virus fixe et qui précède d'un jour les manifestations nerveuses de la rage. Il semble donc justifié de regarder les faibles élévations de température dans les premières semaines après la morsure, comme des signes de mauvais augure.

*Différentes méthodes de vaccination antirabique.*—Après ces recherches préliminaires, nous sommes entré dans l'étude de la vaccination antirabique proprement dite, recherches poursuivies avec tant de succès par M. Pasteur et ses collaborateurs. Ces recherches s'imposent tout de même, surtout en considérant deux circonstances :—

(1°.) Les insuccès qu'on observe encore parfois d'après le procédé de Pasteur ;

(2°.) La virulence de la substance vaccinatoire. Malgré les résultats incontestables obtenus par le méthode Pasteur, il est donc justifié de chercher à perfectioner le procédé.

Pour y arriver j'ai essayé tout d'abord d'assurer le passage lent des moëlles inoffensives aux moëlles virulentes, en j'avais en effet publié en 1886 (*Connaisances médicales*) des résultats très favorables obtenus sur le chien par un mélange des moëlles et par la répétition des séries d'inoculation. Une modification dans ce sens a été introduite aussi par M. Pasteur dans son traitement antirabique.

J'ai essayé ensuite de vacciner des chiens avec des grandes quantités de moëlle à la limite de sa virulence, et nous avons réussi en effet de

vacciner même contre l'effet de l'infection intracranienne avec des moëlles de six jours. Cependant nous avons constaté en même temps que l'inoculation des très grandes doses de substance rabique est parfois toxique et amène la mort des animaux avec marasme et parfoit avec une nephrite.

Comme j'avais constaté que la température de 60° C. détruit la faculté rabigène de la moëlle infectieuse j'ai essayé de vacciner les chiens avec la substance rabique chauffée à 80 C., c'est-à-dire, inoffensive. On réussit en effet à donner l'immunité par des grandes quantités de cette substance, cependant il faut habituer l'organisme à ces grandes quantités en commençant par l'injection de quantités croissantes.

On arrive aussi à vacciner les chiens par des séries des moëlles exposées à une température de 50–60° C., ou bien par l'inoculation du virus dilué. Cependant ces derniers procédés ne sont pas tout à fait inoffensifs et n'ont pas d'avantage sur la vaccination d'après Pasteur.

Pourtant il fallait chercher une méthode perfectionnée pour prévenir les suites des morsures terribles des loups enragés, pour lesquelles la méthode Pasteur ne donne pas toujours des bons résultats.

Au commencement en pratiquant chez les mordus le traitement fort de Pasteur, nous avions plusieurs insuccès; il nous fallait employer une méthode beaucoup plus forte, méthode qui s'impose chez nous, aussi à cause de nos petits lapins dont la moëlle est moins efficace.

Nous donnons donc à ces mordus toute la série des moëlles en trois jours et nous répétons l'inoculation de la série 6–8 fois pendant 30 jours en allant un à deux fois jusque à l'inoculation des moëlles fraîches. Un fait de grande gravité est d'une part l'arrivée relativement tard des mordus par des loups et d'autre part la rapidité avec laquelle la maladie se déclare.

Ainsi serait-il impossible de prévenir la maladie dans des cas où elle se déclare 14 jours après la morsure, ce qui arrive assez souvent après les morsures terribles du loup.

Il s'agit dans ces cas d'arriver aussi vite que possible à un vaccin efficace.

Ces considérations en même temps que celles que j'ai énumérées plus haut, m'ont conduit à des recherches étendues sur l'effet vaccinatoir du sang et des liquides des auimaux refractaires à la rage.

J'avais constaté que la grenouille ne contracte pas la rage, même is on la tient à une température de 30–37° C. Il était donc important de voir si les liquides organisés de cet animal ne sont pas vaccinatoirs.

M. A. Babes avait isolé les albumoses des organes de cet animal et il avait essayé de vacciner des chiens et des lapins avec ces substances sans y parvenir. Nous avons ensuite essayé l'effet des liquides vivants pour y arriver, et nos essais ont eu le résultat que la substance rabique est atténué lentement dans le sac lymphatique dorsal de la grenouille et que par cette substance atténuée on peut vacciner des chiens. La lymphe de la grenouille, melée avec la substance rabique, détruit son effet

rabigène en cinq à six heures. Cependant ces faits ne sont pas applicables à la vaccination antirabique.

Un autre procédé, au contraire, dont l'efficacité m'avait déjà frappé il y a deux ans et dont j'avais publié les résultats (Ann. de l'Inst. Pasteur 1889), semblait propre au perfectionnement du traitement antirabique.

C'est l'injection systématique du sang des animaux refractaires et surtout de celui du chien vacciné et révacciné. Nous avons employé dans ce but des chiens de grande taille, qui après la vaccination ont été infectés par la voie intracranienne, d'abord avec le virus des rues et après un nouveau traitement très abondant avec le virus fixe. Une seule inoculation ne suffit pas, mais si on injecte pendant huit jours, chaque jour cinq grammes de sang à des chiens sains ou infectés par morsure, ou même par trépanation, on constate que les animaux deviennent par ces inoculations, refractaires à une infection ultérieure et même antérieure. Même après avoir introduit le virus des rues par trépanation, nous sommes arrivés à guérir dans une série quatre sur dix, dans une autre deux sur quatre, et dans une troisième série un sur trois chiens. Chez les chiens où la rage éclatait malgré ce traitement, elle a été ordinairement beaucoup retardée. Il va sans dire que l'effet de ces inoculations a toujours été controlé et que les animaux d'épreuve sont toujours morts de la rage.

*Preuves pour l'efficacité du traitement antirabique.*—Sans entrer dans des détails je terminerai cette communication en donnant quelques preuves tirées de nos expériences pour l'efficacité du traitement antirabique. Quoique les statistiques antérieures des vaccinations Pasteuriennes ne sont pas sans reproche, elles montrent tout de même d'une façon évidente l'efficacité du traitement. Cependant nous étions dans la position de pouvoir apporter des preuves qui se rapprochent beaucoup des preuves expérimentelles, à savoir :—

(1.) Sur 1,100 personnes mordues par des chiens enragés et qui ont pu terminer le traitement, nous avons obtenu un résultat qui reste au dessous de cinq pro mille, tandis que parmi 25 personnes qui dans la même époque sont venues à notre institut, mais qui se sont soustraites au traitement et qui ont été tenues en surveillance, cinq sont mortes, c'est-à-dire, 20 pour cent. Dans trois de ces cas le même chien avait mordu plusieurs personnes, et les personnes soumises au traitement ont survécu.

(2.) Avant l'inauguration du traitement antirabique 60 °/₀ environ des personnes mortes de la rage, ont succombé entre le 45 et 75 jours après la morsure, tandis que seulement 20 °/₀ sont mortes dans le premier mois après la morsure. Le traitement antirabique qui commence son action préservative énergique seulement 14 jours après la fin du traitement n'est pas donc très efficace pour empêcher la manifestation de la rage dans le premier mois après la morsure, et on voit en effet que parmi les insuccès, à peu près 60 °/₀ reviennent au

premier mois après la morsure, tandis que parmi les insuccès après vaccinations ce sont à peine 20 °/₀ qui reviennent aux 45e–75e jours après la morsure. On peut donc calculer que sans vaccination dans cette époque au lieu d'un pour mille, la mortalité aurait été au moins huit pour mille.

(3.) D'après notre statistique antérieure au traitement Pasteurien, 90 °/₀ des personnes mordues à la face par des loups enragés, meurent de la rage.

Par un traitement trop faible on perd à peu près que moitié des mordus gravement à la face par des loups enragés, tandis que par un traitement très fort, nous n'en perdons plus que 12 °/₀ qui meurent pendant le traitement et à peine 2 °/₀ parmi les personnes qui ont pu finir traitement.

(4.) Parmi 12 personnes mordus à Budesti par un loup enragé, une seule est morte de la rage, tandis que les autres ont été sauvées par un traitement très énergique, tandis que tous les 31 animaux domestiques (chevaux, bœufs, porcs, et chiens) mordus en même temps que les hommes, ont succombé de la rage.

(5.) Parmi 31 personnes atteintes par le même loup enragé près de Cernovitz, cinq ont été simplement égratignées, une personne mordue au bras à travers les vêtements a été traitée à Budapest, une seule femme gravement mordue est restée à Cernovitz, et tous les autres, c'est-a-dire, 25 personnes, terriblement mordues à la face, sont venues à Bucarest pour se faire traiter. Parmi ces personnes cinq sont venues seulement 10 jours après la morsure. Chez une de ces dernières la rage éclatait quatre jours après le commencement du traitement. Les deux personnes les plus grièvement mordues, mouraient de la rage, pendant le traitement, en moins de 30 jours après la morsure, mais toutes les personnes qui ont pu finir le traitement sont bien portantes et seulement la personne mordue, qui n'avait pas été soumise au traitement antirabique, est morte de la rage.

Ce dernier cas, tellement démonstratif, nous a servi en même temps pour des études sur l'efficacité du traitement antirabique de l'homme avec le sang des hommes et des animaux refractaires à la rage.

Après avoir établi l'efficacité de ce traitement inoffensif pour le chien, rien ne s'opposait pas à son emploi chez l'homme. C'était surtout l'état désespéré de ces mordus et le résultat incomplet, même d'un traitement très énergique, qui m'avaient inspiré.

J'ai donc divisé les mordus en deux groupes égaux, les plus gravement mordus ont reçu en même temps que le traitement Pasteur, de deux en deux jours, d'injections alternatives (10 grammes) de sang d'hommes et de chiens immunisés, et en effet le résultat de ce traitement a été très encourageant, car une seule des personnes plus gravement mordues et qui avait reçu du sang, a uccombé de la rage, tandis que parmi les personnes, moins gravement mordues et qui n'ont pas reçu du sang, deux sont mortes de la rage.

Depuis j'ai practiqué plusieurs fois cette vaccination tout à fait inoffensive, surtout dans des faits où les personnes gravement mordues et arrivées tard la désirent. Dans ces cas je donne pendant les premiers jours et à la fin du traitement Pasteurien, chaque jour 10 grammes de sang, et je suis persuadé que ce traitement efficace rendra des bons services dans les cas où il est de la plus grande importance d'arriver si vite que possible à des vaccins.

J'espère que ces recherches contribueront à élargir la base expérimentale sur laquelle repose le traitement antirabique, et que mes observations sur l'homme donneront des preuves de la valeur des expériences pour l'efficacité du traitement antirabique.

## Ueber die praktischen Erfolge der antirabischen Schutzimpfungen in Budapest während des ersten, vom 15. April 1890—14. April 1891, sich erstreckenden Jahres.

VON

Prof. Dr. A. Högyes, Budapest.

[This Paper was originally contributed to Section II.]

### *I.—Einleitende Bemerkungen.*

Nach vorangehenden, langwährenden Thierexperimenten begann ich die antirabischen Impfungen am Menschen am 15. April 1890 in einem zu diesem Zwecke eingerichteten provisorischen Laboratorium, in Verbindung mit meinem Institut für Allgemeine Pathologie und Therapie. Die Einrichtung des Laboratoriums und des Dienstes ist im Grossen und Ganzen dem Pariser Pasteurschen Institut für antirabische Schutzimpfungen entsprechend, nur sind meine Localitäten viel kleiner und weniger geeignet als letztere. Die Schutzimpfungen geschehen kostenlos. Die Ausgaben des Institutes werden von einem, durch das Parlament votirten Budget gedeckt. Die von wuthkranken Hunden gebissenen Armen des Landes geniessen ausserdem noch die Begünstigung, dass sie an den Staatseisenbahnen nach Budapest und zurück Kostenfrei reisen können, und dass sie ferner in den Spitälern während der Dauer der Behandlung gratis verpflegt werden. Durch diese Vorkehrungen ist es ermöglicht, dass die von wuthkranken Hunden Gebissenen des Landes zum grössten Theil und genügend schnell den antirabischen Schutzimpfungen sich unterwerfen können.

Nach der Beendigung der Behandlung wird im 3. oder 4. Monat über den Gesundheitszustand eines jeden behandelten Individuums eine ämtliche Erkundigung eingezogen, wodurch es ermöglicht ist über die Resultate der Schutzimpfungen eine möglichst getreue Statistik zusammenzustellen.

Da nach der Beendung der erstjährigen Impfungen (14. April 1891) die drei Monate soeben verstrichen sind und sämmtliche ämtliche

Erkundigungen über die zuletzt Behandelten noch nicht erledigt sind, so sind die mitzutheilenden statistischen Daten nur approximativ, obschon sie voraussichtlich keine Aenderung erfahren werden, da die Todesfälle, wenn auch nicht immer, so doch in den meisten Fällen früher als die ämtlichen Meldungen erfahren werden, und da es ein seltener Fall ist, dass an dem Geimpften die Wuthkrankheit nach 3 Monaten ausbreche.*

## *II.—Statistische Daten.*

In dem oben bezeichneten Zwischenraum eines Jahres erfuhren insgesammt 701 Individuen die antirabische Behandlung.

Die Fälle kann man, nach der statistischen Zusammenstellungs-Methode des Pasteurschen Institut in folgende statistische Tabelle fassen :—

DIE STATISTIK DER BUDAPESTER ANTIRABISCHEN SCHUTZIMPFUNGEN vom 15. April 1890—14. April 1891.

| — | | A. | | | B. | | | C. | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Bemerk. | Einzelne Fälle. | Summe. | Bemerk. | Einzelne Fälle. | Summa | Bemerk. | Einzelne Fälle. | Summe. |
| *Kopf- und Gesichtswunde* | Einzelne | . | . | 2 | . | 7 | 55 | . | 5 | 15 |
| | Mehrere | . | 2 | | . | 48 | | . | 10 | |
| Genügende Ausätzung | | . | . | . | . | . | . | . | . | . |
| Ungenügende Ausätzung | | 1 | . | . | 34 | . | . | 6 | . | . |
| Ausätzung geschah nicht | | 1 | . | . | 21 | . | . | 9 | . | . |
| *Handwunde* | Einzelne | . | 19 | 43 | . | 46 | 186 | . | 10 | 35 |
| | Mehrere | . | 24 | | . | 140 | | . | 25 | |
| Genügende Ausätzung | | 1 | . | . | 5 | . | . | 1 | . | . |
| Ungenügende Ausätzung | | 23 | . | . | 101 | . | . | 25 | . | . |
| Ausätzung geschah nicht | | 19 | . | . | 80 | . | . | 9 | . | . |
| *Wunden an Extremitäten und Rumpf* | Einzelne | . | 4 | 31 | . | 69 | 237 | . | 15 | 64 |
| | Mehrere | . | 27 | | . | 168 | | . | 49 | |
| Genügende Ausätzung | | . | . | . | 1 | . | . | 1 | . | . |
| Ungenügende Ausätzung | | 13 | . | . | 136 | . | . | 43 | . | . |
| Ausätzung geschah nicht | | 18 | . | . | 100 | . | . | 20 | . | . |
| Gewand durchrissen | | 14 | . | . | 114 | . | . | 39 | . | . |
| Gewand nicht durchrissen | | 9 | . | . | 46 | . | . | 14 | . | . |
| Am nackten Körper | | 8 | . | . | 87 | . | . | 11 | . | . |

* Diese statistische Daten können als definitiv betrachten werden, weil bis heute unter den behandelten Personen sind keine neuere Todesfälle vorgekommen, Budapest, III. 27, 1892.

| — | A. | | | B. | | | C. | | |
|---|---|---|---|---|---|---|---|---|---|
| | Bemerk. | Einzelne Fälle. | Summe. | Bemerk. | Einzelne Fälle. | Summe. | Bemerk. | Einzelne Fälle. | Summe. |
| *Mehrere Wunden an verschiedenen Körperstellen* - - - | . | 2 | 2 | . | 24 | 24 | . | 7 | 7 |
| Genügende Ausätzung - - | . | . | . | . | . | . | . | . | . |
| Ungenügende Ausätzung - - | 2 | . | . | 17 | . | . | 4 | . | . |
| Ausätzung geschah nicht - - | . | . | . | 7 | . | . | 3 | . | . |
| Gewand durchrissen - - | 2 | . | . | 14 | . | . | 4 | . | . |
| Gewand nicht durchrissen - - | . | . | . | 2 | . | . | 1 | . | . |
| Am nackten Körper - - | . | . | . | 8 | . | . | 2 | . | . |
| | A=78. | | | B=502. | | | C=121. | | |
| | | | | Totalsumme=701. | | | | | |

Columne A. enthält jene gebissenen Personen, welche von nehsolc Thieren gebissen wurden, deren Wuthkrankheit experimentell festgestellt ist; Columne B. solche, deren Wuthkrankheit der Thierarzt constatirte; Columne C. solche Personen, welche von wuthverdächtigen Thieren inficirt wurden.

Das inficirende Thier war: in 601 Fällen Hunde, in 85 Fällen Katzen, in 8 Fällen Kühe, in 2 Fällen Esel, in 2 Fällen Pferde, in 2 Fällen Schweine, in 1 Falle ein Kalb.

Von diesen 701 Personen starben bisher (15. Juli 1891) 20 Individuen an der Wuthkrankheit.

### *III.—Bestimmung des Mortalitäts-Verhältnisses aus obigen Angaben.*

Indem von den behandelten 701 Personen 20 Individuen verstarben an der Wuthkrankheit, so ist die gesammte Mortalität 2·7%.

Von diesen 20 Personen aber verstarben: 1 während der Behandlung; 11 nach der Behandlung innerhalb 15 Tage; 8 Personen um 15 Tage später nach der Behandlung.

Bei der Bestimmung des wirklichen Mortalitäts-Verhältnisses kann man nur letztere Zahl als massgebend betrachten, da für jene Personen welche während der Behandlung sowie in den ersten 2 Wochen nach der Behandlung verstarben, nach Pasteurs Auseinandersetzungen anzunehmen ist, dass das Virus der Strassen-Wuthkrankheit schon am Anfang der Behandlung oder während letzterer zu den nervösen Centren des inficirten Individuums gelangte, und somit für solche Personen die immunisirende Wirkung der Schutzimpfungen bereits als verspätet erscheint.

Diese Annahme basirt sich auf jene experimentelle Erfahrung, laut welcher ein Hund, welcher mit dem Virus der Strassen-Wuthkrankheit mittelst Trepanation subdural inficirt wurde, zwischen dem 14. und 20.

Tag erkrankt. Meine eigenen experimentellen Erfahrungen sprechen auch hierfür.

Somit erscheint es motivirt, dass wir von den verstorbenen 20 Personen die ersten 12 aus der Rechnung ausschliessen, und nur die letzten 8 Todesfälle als solche betrachten, in welchen der Tod trotz der Anwendung der antirabischen Behandlung eintraf.

Nach dieser Reduction ist die statistische Thatsache und auf deren Grund das Mortalitäts-Verhältniss folgend:—

Subtrahirt man von den 701 Fällen die eliminirten 12 Fälle, so restiren 689. Von diesen 689 Fällen starben insgesammt 8.

Dies entspricht einem Mortalitäts-Verhältniss von 1·1%.

### *IV.—Das Mortalitäts-Verhältniss bei den verschiedenen Schutzimpfungs-Methoden.*

Von obigen 689 Individuen vollzog ich an 299 die antirabischen Schutzimpfungen nach Pasteurs ursprünglicher classischer Methode mit dem getrockneten Rückenmarke von wuthkranken Passage-Kaninchen, hauptsächlich nach der sog. intensiveren Behandlungsweise.

Bei den übrigen 390 wandte ich zu Schutzimpfungen jenes Verfahren an, welches ich zur Immunisirung der Hunde und als präventives Verfahren gegen den Ausbruch der Wuthkrankheit nach einem Biss von wuthkranken Hunden mit Erfolg gebrauchte.*

Dieses Verfahren besteht darin, dass ich aus der ganz frischen und ungetrockneten Oblongata eines Passage-Kaninchen mit einer (7-promilligen) 7‰ sterilisirten Kochsalzlösung verschiedene Dilutionen verfertigte (1:100, 1:200, 1:300, 1:500, 1:2000, 1:5000, 1:8000, 1:10,000) und injicirte erst die schwächeren, hernach die stärkeren Dilutionen successive subcutan. Mit diesen successiven Injectionen kann man den Organismus langsam bis zur am stärksten wirkenden Dilution (1:200, 1:100) gewöhnen, so dass mit entsprechender Vorsicht in der Dosirung bis zu immer stärkeren Lösungen gestiegen, die Gefahr vollkommen ausgeschlossen ist, wie ich dies in vorhinein an 70 Hunde ausprobirte, ohne dass auch nur einem Etwas dabei geschehen wäre, während, wie bekannt, die concentrirten Dilutionen des Passage-Markes nicht ohne Gefahr unter die Haut zu injiciren sind; der grösste Theil der somit inficirten Hunde bleibt zwar am Leben und wird immun, ein Theil jedoch acquirirt auf diese Art die Wuthkrankheit. Dieses Risico entfällt bei obigem Verfahren vollkommen.

Die Marktrocknung attenuirt die Virulenz des frischen Markes gleichfalls dadurch, dass sie die Menge des Wuth-Virus von Tag zu Tag in dem trocknenden Marke vermindert. Experimente an Kaninchen zeigten, dass die schwächeren Dilutionen des frischen Passage-Virus subdural injicirt ebenso wenig das Thier tödten, wie die Emulsionen des 12–14 Tage getrockneten Passage-Rückenmarks, während die subdural applicirten stärkeren Dilutionen des frischen Marks das

---

* Siehe *A. Höyes:* Die experimentelle Basis der Pasteurschen Antirabischen Schutzimpfungen. Enke, Stuttgart, 1890.

Kaninchen bereits wuthkrank machen, gleichwie das einige Tage getrocknete Kaninchenmark, und zwar nach umso kürzerer Incubation, je stärkere Dilution wir anwandten, ebenso wie die Emulsionen aus getrocknetem Mark das Thier umso schneller tödten, aus je kürzer getrocknetem Mark wir dieselbe anfertigen.

Der Umstand einestheils, dass das Verfahren bei entsprechender Vorsicht unschädlich ist, anderntheils, dass ich bei meinen Thierexperimenten Pasteurs grundlegende Thierexperimente wiederholend zu günstigere Resultate gelangte als bei Schutzimpfungen mit getrockneten Mark: ermunterte mich, dass ich mein Verfahren an, von wuthkranken Thieren inficirten Personen versuche.

Der Erfolg entsprach denn auch der Erwartung.

Das Verfahren erwies sich einestheils auch beim Menschen für unschädlich, anderntheils schien auch das Mortalitäts-Verhältniss sich günstiger zu gestalten. Von den, mit Emulsion aus getrocknetem Mark geimpften 299 Personen wurden 5 wuthkrank, was einer Mortalität von 1·67% entspricht; von den, mit Dilutionen aus frischem Wuthmark geimpften 390 Personen starben 3, was einer Mortalität von 0·67% gleichkommt.

Ich bin geneigt, diese beim Menschen erreichte günstigeren Erfolge mit Markdilutionen auf denselben Umstand zurückzuführen, welchen ich bei meinen Thierexperimenten erörterte. Die verschieden dicke Rückenmarke der verschieden grossen Kaninchen trocknen im ungleichen Verhältniss, die Verminderung der Virus-Quantität ist von Tag zu Tag nicht gleichmässig. Daher ist die Successivität der Virus-Injection in der Form von Emulsion aus getrocknetem Mark nicht so exact, wie wenn man das frische Mark abwägt und entsprechend diluirt.

Im Pariser Pasteur-Institut werden zu den Schutzimpfungen gleichartige und gleich grosse Kaninchen gebraucht, deren Rückenmark ziemlich gleich dick ist und gleichmässig trocknen mag; die aus denselben angefertigte Markemulsion enthält denn auch das Virus in gleichmässiger Successivität, womit die Dosirung auch genügend verlässlich erscheint.

In solchen Instituten, wo zu diesem Zwecke die Einrichtung der Züchtung einer besonderen Kaninchenart mangelt, wo daher kleinere und grössere Kaninchen gemengt gebraucht werden zur Herstellung des fixen Virus: dort erscheint zur genauen Dosirung des, als Vaccine dienenden Marks die Methode der Dilution zweckentsprechend, wofür ausserdem noch die einfache Art der Anfertigung spricht.

Obige Zahlen sind zum Vergleich noch zu gering. Ich werde die Frage weiter studiren.

### *V.—Mortalitäts-Verhältniss zwischen den geimpften und nicht geimpften Gebissenen von wuthkranken Thieren.*

Bei der Beurtheilung der Efficacität der antirabischen Schutzimpfungen ist zweckmässig der Vergleich des Mortalitäts-Verhältnisses der, unter derselben Zeit Geimpften und Nicht-Geimpften. Dieses comparative Vorgehen gebrauchten *Dujardin-Baumetz* bei der Zusam-

menstellung der in antirabischer Behandlung gestandener und letzterer nicht theilhaftig gewordener Gebissenen von wuthkranken Thieren des Seine-Departement, und *Sir Roscoe* bei jener von England. Dasselbe Vorgehen wandte ich für die, im Pariser Pasteur-Institut behandelten und nicht-behandelten ungarländischen Inficirten.

Aus diesen drei Zusammenstellungen erhellte, dass das Mortalitäts-Verhältniss der ohne antirabischer Behandlung Gebliebenen 9·3°/₀–15°/₀ war, während dieses bei Pasteursche Impfungen Empfangenen unter derselben Zeit 3·3°/₀–1·19°/₀ war.

Im Jahre vom 15. April 1890—14. April 1891 starben von 689, im Budapester antirabischen Institute behandelten Personen—wie wir oben sahen—trotz der Schutzimpfungen, 8 Individuen. Ich weiss zwar nicht, wie viel, unter derselben Zeit, von wuthkranken Hunden gebissenen Personen einer antirabischen Behandlung nicht unterworfen waren, da wir die ämtlichen Daten eben jetzt wollen sammeln, wir wissen jedoch schon dieser Zeit, dass von diesen bereits 20 starben;* ja wenn wir die obigen 12 Todesfälle auch hinzurechnen, so steigt diese Zahl auf 30; und es ist doch sehr wahrscheinlich, dass in Anbetracht der oben erwähnten staatlichen Begünstigungen, welche auch den Aermsten leicht ermöglichen sich den Schutzimpfungen zu unterwerfen, die Zahl der zur Behandlung nicht erschienenen Inficirten verschwindend klein sein wird gegen jene der erschienenen, und antirabischen Wuthbehandlung unterworfenen, von wüthenden oder wuthverdächtigen Thieren gebissenen Personen.

## Résultats statistiques de l'Institut Antirabique d'Odessa.—18 tableaux.

PAR

le Dr. J. Bardach, Directeur, Institut Pasteur, Odessa.

[This Paper was originally contributed to Section II.]

Je me permets de présenter une statistique des résultats du traitement préventif de la rage. Nos observations faites pendant une période de quatre années (1887–90) à l'Institut Bactériologique d'Odessa permettent de se rendre compte de l'éfficacité de la méthode Pasteur. Pendant cette période 2,243 personnes ont subi le traitement; 16 entre elles sont mortes. Ces chiffres montrent avec évidence, combien est faible la mortalité après traitement: 0·72 pour cent. Si selon la statistique de l'Institut Pasteur, nous prenons seulement pour mettre en évidence les résultats obtenus dans les cas où la mort se produit après deux semaines, à compter de la fin du traitement, nous obtenons les résultats suivants: personnes traitées, 2,238; décès, 11; mortalité pour cent., 0·49. Ces résultats, obtenus dans un laps de quatre années sur plus de deux mille cas, peuvent déjà indiquer la valeur de la méthode, d'autant plus qu'ils

* Diese Zahl der Todesfälle unter den nicht behandelten Personen stiegt bisher (III. 26, 1892) nach den ämtlichen Gerichten 34 auf.

sont en concordance avec les chiffres qui nous viennent d'être fournis par l'Institut Pasteur.

Il faut pourtant ajouter que la puissance de la méthode a de certaines limites. Le taux actuel des insuccès peut être exprimé par un chiffre. En effet, si nous prenons les cas où les sujets se sont présentés dans la première semaine, à dater de la morsure, nous verrons que sur 1,659 cas il y a néanmoins 14 décès, soit 0·84 pour cent. Nous en trouverons l'explication dans ce fait suivant :—les morsures sont alors multiples, très graves, très rapprochées des centres nerveux, et, par conséquent, la période d'incubation est très courte. Mais cette cause n'est pas la seule. Il faut tenir compte des différences individuelles qui doivent jouer dans ce cas un rôle important; car il y a des morsures multiples, extrêmement graves et très rapprochées du cerveau, qui néanmoins à la suite de traitement sont guéris.

Analysons, maintenant, en détail les résultats obtenus, et étudions d'abord les morsures à la figure et à la tête, qui sont à juste titre considérées comme les plus dangereuses. Dans ces parties le réseau nerveux étant très resserré, cela rend le voisinage du cerveau plus dangereux encore, en facilitant l'arrivée déjà si prompte du microbe dans le centre nerveux. Dès cette arrivée, il se produit dans les ganglions des modifications rapides qui entraînent la déclaration de la rage. Si nous ne prenons que ceux qui ont subi le traitement jusqu'à la fin, nous voyons que le nombre des décès est de trois sur 198, soit 1·51 pour cent. La mortalité chez les personnes mordues aux mains s'exprime par le chiffre suivant: cas traités, 1,386; décès, 9; mortalité pour cent. 0·69. Passons maintenant au groupe des mordues au membre et au tronc. Nous trouvons dans ce groupe: cas traités, 659; décès, 4; mortalité pour cent., 0·61.

Des faits exposés je crois pouvoir conclure que la méthode est éfficace; les décès sont très rares, même dans les cas les plus graves, ce qui prouve que la méthode du traitement anti-rabique, due au génie de M. Pasteur, fondateur de la science bactériologique, est d'une puissance presque absolue.

No. 1.

MORSURES au visage, à la tête, aux mains, aux membres et au tronc.
Mortalité pendant et après le traitement.

| Années. | Tableau A. | | | Tableau B. | | | Tableau C. | | | Total. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Nombre de personnes traitées. | Morts. | Mortalité %. | Nombre de personnes traitées. | Morts. | Mortalité %. | Nombre de personnes traitées | Morts. | Mortalité %. | Nombre de personnes traitées. | Morts. | Mortalité %. |
| 1887 - - | 62 | 0 | 0 | 67 | 1 | 1·49 | 219 | 4 | 1·82 | 348 | 5 | 1·43 |
| 1888 - - | 82 | 2 | 2·43 | 124 | 4 | 3·22 | 241 | 7 | 2·90 | 447 | 13 | 2·90 |
| 1889 - - | 269 | 5 | 1·86 | 231 | 2 | 0·86 | 248 | 4 | 1·61 | 748 | 11 | 1·47 |
| 1890 - - | 211 | 3 | 1·42 | 261 | 4 | 1·53 | 251 | 3 | 1·19 | 723 | 10 | 1·38 |
| Totaux - | 624 | 10 | 1·60 | 683 | 11 | 1·61 | 959 | 18 | 1·87 | 2,266 | 39 | 1·72 |

## No. 2.

**Morsures au visage, à la tête, aux mains, aux membres et aux tronc.**
**Mortalité après les traitement.**

| Années. | Tableau A. | | | Tableau B. | | | Tableau C. | | | Total. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. |
| 1887 - - | 62 | 0 | 0 | 67 | 1 | 1·49 | 216 | 1 | 0·46 | 345 | 2 | 0·58 |
| 1888 - | 81 | 1 | 1·23 | 121 | 1 | 0·82 | 237 | 3 | 1·27 | 439 | 5 | 1·14 |
| 1889 - - | 265 | 1 | 0·37 | 230 | 1 | 0·43 | 246 | 2 | 0·81 | 741 | 4 | 0·54 |
| 1890 - - | 211 | 3 | 1·42 | 258 | 1 | 0·38 | 249 | 1 | 0·40 | 718 | 5 | 0·69 |
| Totaux - | 619 | 5 | 0·80 | 676 | 4 | 0·59 | 948 | 7 | 0·73 | 2,243 | 16 | 0·71 |

## No. 3.

**Morsures au visage à la tête, aux mains, aux membres et au tronc.**
**Mortalité 15 jours après le traitement.**

| Années. | Tableau A. | | | Tableau B. | | | Tableau C. | | | Total. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. |
| 1887 - - | 62 | 0 | 0 | 67 | 1 | 1·49 | 216 | 1 | 0·46 | 345 | 2 | 0·58 |
| 1888 - - | 81 | 1 | 1·23 | 121 | 1 | 0·82 | 236 | 2 | 0·85 | 438 | 4 | 0·91 |
| 1889 - - | 265 | 1 | 0·37 | 230 | 1 | 0·43 | 245 | 1 | 0·40 | 740 | 3 | 0·40 |
| 1890 - - | 208 | 0 | 0 | 258 | 1 | 0·38 | 249 | 1 | 0·40 | 715 | 2 | 0·28 |
| Totaux - | 616 | 2 | 0·32 | 676 | 4 | 0·59 | 946 | 5 | 0·52 | 2,238 | 11 | 0·49 |

## No. 4.

**Morsures au visage et à la tête.**
**Mortalité pendant et après le traitement.**

| Années. | Tableau A. | | | Tableau B. | | | Tableau C. | | | Total. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. |
| 1887 - - | 6 | 0 | 0 | 6 | 0 | 0 | 14 | 2 | 14·28 | 26 | 2 | 7·69 |
| 1888 - - | 9 | 1 | 11·11 | 15 | 3 | 20·00 | 27 | 4 | 14·81 | 51 | 8 | 15·68 |
| 1889 - - | 25 | 4 | 16·0 | 21 | 1 | 4·76 | 27 | 3 | 11·11 | 73 | 8 | 10·95 |
| 1890 - - | 14 | 0 | 0 | 28 | 0 | 0 | 24 | 3 | 12·50 | 66 | 3 | 4·54 |
| Totaux - | 54 | 5 | 9·25 | 70 | 4 | 5·71 | 92 | 12 | 13·04 | 216 | 21 | 9·27 |

## No. 5.

**Morsures au visage et à la tête.**

**Mortalité après le traitement.**

| Années. | Tableau A. | | | Tableau B. | | | Tableau C. | | | Total. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. |
| 1887 - - | 6 | 0 | 0 | 6 | 0 | 0 | 12 | 0 | 0 | 24 | 0 | 0 |
| 1888 - - | 8 | 0 | 0 | 13 | 1 | 7·69 | 23 | 0 | 0 | 44 | 1 | 2·27 |
| 1889 - - | 21 | 0 | 0 | 20 | 0 | 0 | 25 | 1 | 4·00 | 66 | 1 | 1·51 |
| 1890 - - | 14 | 0 | 0 | 28 | 0 | 0 | 22 | 1 | 4·54 | 64 | 1 | 1·56 |
| Totaux - | 49 | 0 | 0 | 67 | 1 | 1·49 | 82 | 2 | 2·44 | 198 | 3 | 1·51 |

## No. 6.

**Morsures aux mains.**

**Mortalité pendant et après le traitement.**

| Années. | Tableau A. | | | Tableau B. | | | Tableau C. | | | Total. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. |
| 1887 - - | 44 | 0 | 0·00 | 40 | 1 | 2·50 | 134 | 2 | 1·49 | 218 | 3 | 1·38 |
| 1888 - - | 50 | 1 | 2·00 | 75 | 1 | 1·33 | 121 | 3 | 2·48 | 246 | 5 | 2·03 |
| 1889 - - | 184 | 1 | 0·54 | 148 | 1 | 0·67 | 139 | 1 | 0·72 | 471 | 3 | 0·63 |
| 1890 - - | 144 | 0 | 0·00 | 153 | 2 | 1·31 | 158 | 0 | 0·00 | 455 | 2 | 0·44 |
| Totaux - | 422 | 2 | 0·47 | 416 | 5 | 1·20 | 552 | 6 | 1 08 | 1,3 0 | 13 | 0·93 |

## No. 7.

**Morsures aux mains.**

**Mortalité après le traitement.**

| Années. | Tableau A. | | | Tableau B. | | | Tableau C. | | | Total. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. |
| 1887 - - | 44 | 0 | 0 | 40 | 1 | 2·5 | 133 | 1 | 0·75 | 217 | 2 | 0·92 |
| 1888 - - | 50 | 1 | 2·0 | 74 | 0 | 0 | 121 | 3 | 2·48 | 245 | 4 | 1·63 |
| 1889 - - | 184 | 1 | 0·54 | 148 | 1 | 0·67 | 139 | 1 | 0·72 | 471 | 3 | 0·63 |
| 1890 - - | 144 | 0 | 0 | 157 | 0 | 0 | 158 | 0 | 0 | 453 | 0 | 0 |
| Totaux - | 422 | 2 | 0·47 | 413 | 2 | 0·48 | 551 | 5 | 0·90 | 1,386 | 9 | 0·65 |

## No. 8.

**Morsures aux membres et au tronc.**

**Mortalité pendant et après le traitement.**

| Années. | Tableau A. | | | Tableau B. | | | Tableau C. | | | Total. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. |
| 1887 - - | 12 | 0 | 0 | 21 | 0 | 0 | 71 | 0 | 0 | 104 | 0 | 0 |
| 1888 - - | 23 | 0 | 0 | 34 | 0 | 0 | 93 | 0 | 0 | 150 | 0 | 0 |
| 1889 - - | 60 | 0 | 0 | 62 | 0 | 0 | 82 | 0 | 0 | 204 | 0 | 0 |
| 1890 - - | 53 | 3 | 5·66 | 80 | 2 | 2·5 | 69 | 0 | 0 | 202 | 5 | 2·47 |
| Totaux - | 148 | 3 | 2·02 | 197 | 2 | 1·01 | 315 | 0 | 0 | 660 | 5 | 0·75 |

## No. 9.

**Morsures aux membres et au tronc.**

**Mortalité après le traitement.**

| Années. | Tableau A. | | | Tableau B. | | | Tableau C. | | | Total. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. |
| 1887 - - | 12 | 0 | 0 | 21 | 0 | 0 | 71 | 0 | 0 | 104 | 0 | 0 |
| 1888 - - | 23 | 0 | 0 | 34 | 0 | 0 | 93 | 0 | 0 | 150 | 0 | 0 |
| 1889 - - | 60 | 0 | 0 | 62 | 0 | 0 | 82 | 0 | 0 | 204 | 0 | 0 |
| 1890 - - | 53 | 3 | 5·66 | 79 | 1 | 1·26 | 69 | 0 | 0 | 201 | 4 | 1·99 |
| Totaux - | 148 | 3 | 2·02 | 196 | 1 | 0·51 | 315 | 0 | 0 | 659 | 4 | 0·61 |

## No. 10.

**Distribution des Morsures.**

**Mortalité pendant et après le traitement.**

| Années. 1887-90. | Tableau A. | | | Tableau B. | | | Tableau C. | | | Total. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. |
| 1°. Tête et visage | 54 | 5 | 9·25 | 70 | 4 | 5·71 | 92 | 12 | 13·4 | 216 | 21 | 9·72 |
| 2°. Mains - - | 422 | 2 | 0·47 | 416 | 5 | 1·20 | 552 | 6 | 1·8 | 1,390 | 13 | 0·93 |
| 3°. Membres et tronc - | 148 | 3 | 2·02 | 197 | 2 | 1·01 | 315 | 0 | 0 | 660 | 5 | 0·75 |
| Totaux - | 624 | 10 | 1·60 | 683 | 11 | 1·61 | 959 | 18 | 1·87 | 2,266 | 39 | 1·72 |

## No. 11.

### Distribution des Morsures.

Mortalité après le traitement.

| Années. 1887-90. | Tableau A. | | | Tableau B. | | | Tableau C. | | | Total. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. | Nombre de personnes traitées. | Morts. | Mortalité °/₀. |
| 1°. Tête et visage | 49 | 0 | 0 | 67 | 1 | 1·49 | 82 | 2 | 2·43 | 198 | 3 | 1·51 |
| 2°. Mains - - | 422 | 2 | 0·47 | 413 | 2 | 0·48 | 551 | 5 | 0·90 | 1,386 | 9 | 0·65 |
| 3°. Membres et troncs - | 148 | 3 | 2·02 | 196 | 1 | 0·51 | 315 | 0 | 0 | 659 | 4 | 0·61 |
| Totaux - | 619 | 5 | 0·80 | 676 | 4 | 0·59 | 948 | 7 | 0·73 | 2,243 | 16 | 0·71 |

## No. 12.

### Distribution des Morsures.

Sur 2,266 morsures—aux mains—1,330—61·34 °/₀.

Sur 2266 morsures—aux membres et au tronc—660—29·12 °/₀.

Sur 2266 morsures—au visage et à la tête—216—9·53 °/₀.

### Distribution de la Mortalité.

Sur 39 personnes, prises de la rage—pour les morsures au visage et à la tête 21 cas—53·84 °/₀.

Sur 33 personnes, prises de la rage—pour les morsures aux mains 13 cas—33·33 °/₀.

Sur 33 personnes, prises de la rage—pour les morsures aux membres et au tronc 5 cas—12·82 °/₀.

## No. 13.

### Mortalité d'après l'âge.

| —— | Pendant et après le traitement. | Après le traitement. |
|---|---|---|
| Au dessous de 5 ans - | 304 personnes traitées—10 mortes—3·29 °/₀. | 296 personnes traitées—2 mortes—0·67 °/₀. |
| De 5 à 9 ans - - | 452 personnes traitées—6 mortes—1·33 °/₀. | 449 personnes traitées—3 mortes—0·67 °/₀. |
| De 10 à 19 ans - | 517 personnes traitées—7 mortes—1·35 °/₀. | 515 personnes traitées—5 mortes—0·97 °/₀. |
| De 20 à 39 ans - | 681 personnes traitées—9 mortes—1·32 °/₀. | 675 personnes traitées—3 mortes—0·44 °/₀. |
| De 40 à 60 ans - | 245 personnes traitées—5 mortes—2·04 °/₀. | 241 personnes traitées—1 mortes—0·41 °/₀. |
| Plus de 60 ans - - | 67 personnes traitées—2 mortes—2·98 °/₀. | 67 personnes traitées—2 mortes—2·98 °/₀. |

## No. 14.

### La Durée moyenne de l'Incubation.

Pour 39 personnes prises de la rage pendant et après le traitement, 52 jours.

Pour 23 personnes prises de la rage pendant le traitement, 28 jours, (minimum, 12; maximum, 61).

Pour 16 personnes prises de la rage après le traitement, 75 jours, (minimum, 31; maximum, 288).

### La Durée moyenne de l'Incubation.

| | | |
|---|---|---|
| Pour les morsures au visage et à la tête - - | personnes prises de la rage pendant et après le traitement - - | 27 jours |
| | personnes prises de la rage pendant le traitement - - - | 26 jours |
| | personnes prises de la rage après le traitement - - - | 33 jours |
| Pour les morsures aux mains - - - | personnes prises de la rage pendant et après le traitement - - | 61 jours |
| | personnes prises de la rage pendant le traitement - - - - | 37 jours |
| | personnes prises de la rage après le traitement - - - | 72 jours |
| Pour les morsures aux membres et au tronc - | personnes prises de la rage pendant et après le traitement - - | 100 jours |
| | personnes prises de la rage pendant le traitement - - - | 39 jours |
| | personnes prises de la rage après le traitement - - - - | 115 jours |

## No. 15.

Les personnes mordues se présentèrent aux inoculations :—

Pendant la 1er semaine après la morsure dans 1,659 cas, 73·21 %.
Pendant la 2me semaine après la morsure dans 412 cas, 18·18 %.
Pendant la 3me semaine après la morsure dans 98 cas, 4·33 %.
Pendant la 4me semaine après la morsure dans 49 cas, 2·16 %.
Pendant la 5me semaine et plus tard après la morsure dans 48 cas, 2·11 %.

## No. 16.

### 1887–1890.

Moyenne du Chiffre des mordus, traités pendant chacun des mois :—

| | | | |
|---|---|---|---|
| Janvier - - - | 30 | Juillet - - - - | 80 |
| Février - - | 31 | Août - - - | 74 |
| Mars - - - | 42 | Septembre - - | 51 |
| Avril - - - - | 57 | Octobre - - - | 37 |
| Mai - - | 59 | Novembre - - - - | 34 |
| Juin - - - | 69 | Décembre - - - | 21 |

No. 17.

Personnes, inoculées pendant les années 1887, 1888, 1889 et 1890, d'après leur propre désir, sans être mordues, 22.

No. 18.

MORSURES DE LOUPS ENRAGÉS.

| — | Mortalité °/₀. |
|---|---|
| Sur 51 personnes, mordues par des loups enragés sont mortes pendant et après le traitement 10 - - - - | 19·60 °/₀ |
| Sur 43 personnes, mordues par des loups enragés sont mortes après le traitement 2 - - - - | 4·65 °/₀ |

## Sur le Traitement Antirabique de Pasteur.

PAR

le Dr. BORDONI-UFFREDUZZI, Directeur Institut Antirabique, Turin.

[This Paper was originally contributed to Section II.]

L'Institut antirabique du Bureau Municipal d'Hygiène de Turin a commencé à fonctionner le 30 Septembre 1886.

Depuis ce jour jusqu'au 30 Juin 1891, (quatre ans et neuf mois) 1,794, personnes mordues se sont présentées à l'Institut; sur ce nombre 1,344 seulement furent soumises au traitement par la méthode de M. Pasteur; les autres 450 ne furent pas admises parcequ'il avait été démontré que le chien mordeur n'était pas enragé.

Je présente la statistique de notre Institut, dressée d'après la méthode suivie à l'Institut de M. Pasteur à Paris, repartissant en trois catégories A, B, C, les personnes traitées. La première catégorie (A) comprend les personnes mordues par un chien dont la rage a été démontrée expérimentalement, par la mort de l'animal ou par celle d'autres personnes mordues en même temps; la seconde catégorie (B), les personnes mordues par un chien dont la rage a été constatée cliniquement; la troisième (C), les personnes mordues par un animal suspect de rage, c'est-à-dire les cas douteux.

Relativement à la partie du corps sur laquelle portaient les morsures, les personnes soumises au traitement ont été aussi divisées en trois catégories: la première comprend les morsures portant sur la tête; la seconde celles portant sur d'autres parties découvertes (habituellement les mains et les pieds); et enfin la troisième comprend les morsures faites sur les parties couvertes par les vêtements.

Le premier tableau indique le mouvement général de l'Institut depuis le jour de son ouverture jusqu'à la fin du mois de Juin 1891, c'est-à-dire le nombre total des personnes traitées et des morts, répartis par catégories, la mortalité pour chacune de ces dernières et enfin le *quantum* de la mortalité.

Ce dernier chiffre (pour 1,344 personnes traitées) est d'environ 1·40 pour cent.

Notre statistique doit pourtant être répartie en trois périodes correspondantes aux modifications introduites dans le traitement, soit :—

Une première période (2me tableau), du 30 Septembre 1886 au 30 Avril 1887, qui comprend 81 personnes traitées par la méthode appliquée au début par M. Pasteur, légèrement modifiée pour les cas les plus graves.

Une deuxième période (3e tableau), du 1er Mai 1887 au 31 Juillet 1890, qui comprend 925 personnes soumises au même traitement rendu plus intensif par l'augmentation de la quantité de virus inoculé.

Une troisième période (4e tableau) enfin du 1er Août 1890 au 30 Juin 1891, qui comprend 338 personnes traitées par la même méthode rendue encore plus intensive par la quantité de substance inoculée.

L'efficacité de ces modifications, introduites successivement dans la méthode de traitement par M. Pastuer lui-même, est prouvée d'une façon évidente par le taux de la mortalité qui s'est abaissée graduellement jusqu'un chiffre assez bas.

En effet la mortalité qui pour la première période était dans la proportion de 2·46 pour 100, descend dans la deuxième à 1·72 pour 100 et ne dépasse pas 0·29 pour 100 dans la troisième.*

Ces faits sont une nouvelle preuve de la valeur réelle de la *méthode Pasteur* pour le traitement préservatif de la rage, puisque les insuccès deviennent toujours plus rares à mesure que la méthode même le perfectionne.

---

* Cette statistique, faite le 15 Août 1891 sur la demande du comité du Congrès d'hygiène à Londres, n'a pas subi de changements jusqu' à Juin 1892.

## Premier Tableau.

| Années. | Catégories. | Personnes mordues à la tête. | | Personnes mordues sur des parties découvertes. | | Personnes mordues sur des parties couvertes. | | Totaux. | |
|---|---|---|---|---|---|---|---|---|---|
| | | Traités. | Morts. | Traités. | Morts. | Traités. | Morts. | Traités. | Morts. |
| 1886 (3 derniers mois.) | A | 2 | — | 13 | 1 | 2 | — | 17 | 1 |
| | B | 2 | — | 5 | — | 4 | — | 11 | — |
| | C | — | — | 4 | — | 2 | — | 6 | — |
| 1887 | A | 11 | — | 73 | 5 | 34 | — | 118 | 5 |
| | B | 10 | 1 | 54 | — | 22 | — | 86 | 1 |
| | C | — | — | 14 | — | 13 | — | 27 | — |
| 1888 | A | 8 | — | 53 | — | 45 | — | 106 | — |
| | B | 12 | — | 53 | 3 | 72 | — | 137 | 3 |
| | C | — | — | 5 | — | 6 | — | 11 | — |
| 1889 | A | 12 | — | 95 | 1 | 49 | — | 156 | |
| | B | 3 | — | 55 | 2 | 31 | — | 89 | 2 |
| | C | 1 | — | 9 | — | 6 | — | 16 | — |
| 1890 | A | 10 | 2 | 85 | 1 | 44 | — | 139 | 3 |
| | B | 10 | — | 114 | 2 | 67 | — | 191 | 2 |
| | C | 1 | — | 20 | — | 13 | — | 34 | — |
| 1891 (6 premiers mois.) | A | 8 | — | 47 | — | 36 | 1 | 91 | 1 |
| | B | 4 | — | 40 | — | 22 | — | 66 | — |
| | C | 3 | — | 29 | — | 11 | — | 43 | - |
| Totaux | - | 97 | 3 | 768 | 15 | 479 | 1 | 1,344 | 19 |
| Mortalité pour 100 | | 3·03 | | 1·90 | | 0·20 | | 1·40 | |

## Deuxième Tableau.

(Première Période du traitement.)

| Années. | Catégories. | Personnes mordues à la tête. | | Personnes mordues sur des parties découvertes. | | Personnes mordues sur des parties couvertes. | | Totaux. | | Mortalité pour 100. |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Traités. | Morts. | Traités. | Morts. | Traités. | Morts. | Traités. | Morts. | |
| 1886 (6 derniers mois) | A | 2 | — | 13 | 1 | 2 | — | 17 | 1 | — |
| | B | 2 | — | 5 | — | 4 | — | 11 | — | — |
| | C | — | — | 4 | — | 2 | — | 6 | — | — |
| 1887 (4 premiers mois) | | | — | 11 | 1 | 3 | — | 18 | 1 | — |
| | B | 2 | — | 8 | — | 8 | — | 18 | — | — |
| | C | — | — | 7 | — | 4 | — | 11 | — | — |
| Totaux | - | 10 | — | 48 | 2 | 23 | — | 81 | 2 | 2·46 |

## Troisième Tableau.

(Deuxième Période du traitement.)

| Années. | Catégories. | Personnes mordues à la tête. | | Personnes mordues sur des parties découvertes. | | Personnes mordues sur des parties couvertes. | | Totaux. | | Mortalité pour 100. |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Traités. | Morts. | Traités. | Morts. | Traités. | Morts. | Traités. | Morts. | |
| 1887 (8 derniers mois) | A | 7 | — | 62 | 4 | 31 | — | 100 | 4 | — |
| | B | 8 | 1 | 46 | — | 14 | — | 68 | 1 | — |
| | C | — | — | 7 | — | 9 | — | 16 | — | — |
| 1888 | A | 8 | — | 53 | — | 45 | — | 106 | — | — |
| | B | 12 | — | 53 | 3 | 72 | — | 137 | 3 | — |
| | C | — | — | 5 | — | 6 | — | 11 | — | — |
| 1889 | A | 12 | — | 95 | 1 | 49 | — | 156 | 1 | — |
| | B | 3 | — | 55 | 2 | 31 | — | 89 | 2 | — |
| | C | 1 | — | 9 | — | 6 | — | 16 | — | — |
| 1890 (7 premiers mois) | A | 4 | 2 | 60 | 1 | 35 | — | 99 | 3 | — |
| | B | 5 | — | 67 | 2 | 36 | — | 108 | 2 | — |
| | C | — | — | 8 | — | 11 | — | 13 | — | — |
| Totaux | - | 60 | 3 | 520 | 13 | 345 | — | 925 | 16 | 1·72 |

## Quatrième Tableau.

(Troisième Période du traitement.)

| Années. | Catégories. | Personnes mordues à la tête. | | Personnes mordues sur des parties découvertes. | | Personnes mordues sur des parties couvertes. | | Totaux. | | Mortalité pour 100. |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Traités. | Morts. | Traités. | Morts. | Traités. | Morts. | Traités. | Morts. | |
| 1890 (5 derniers mois) | A | 6 | — | 25 | — | 9 | — | 40 | — | — |
| | B | 5 | — | 47 | — | 31 | — | 83 | — | — |
| | C | 1 | — | 12 | — | 2 | — | 25 | — | — |
| 1891 (6 premiers mois) | A | 8 | — | 47 | — | 36 | 1 | 91 | 1 | — |
| | B | 4 | — | 40 | — | 22 | — | 66 | — | — |
| | C | 3 | — | 29 | — | 11 | — | 43 | — | — |
| Totaux | - | 27 | — | 200 | — | 111 | 1 | 338 | 1 | 0·29 |

# Les Résultats des Vaccinations Antirabiques à St. Pétersbourg, du 1/13 Juillet 1886 au 1/13 Janvier 1891.

PAR

le Dr. V. Kraïouchkine, Chef de la Section antirabique de l'Institut Pasteur, St. Pétersbourg.

[This Paper was originally contributed to Section II.]

| Endroit de la Morsure. | 1e Catégorie. | | | 2e Catégorie. | | | 3e Catégorie. | | | Total. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Nombre de Personnes traitées. | Nombre de décès. | Mortalité °/₀. | Nombre de Personnes traitées. | Nombre de décès. | Mortalité °/₀. | Nombre de Personnes traitées. | Nombre de décès. | Mortalité °/₀. | Nombre de Personnes traitées. | Nombre de décès. | Mortalité °/₀. |
| A la tête et à la figure - | 28 | 4 | 10·42 | 10 | 1 | 10 | 14 | 0 | 0 | 52 | 5 | 9·61 |
| Aux mains - - - | 281 | 5 | 1·77 | 132 | 1 | 0·75 | 104 | 5 | 4·80 | 517 | 11 | 2·12 |
| Aux membres et au tronc - | 65 | 0 | 0 | 52 | 0 | 0 | 29 | 0 | 0 | 146 | 0 | 0 |
| Morsures multiples en divers points du corps. | 30 | 4 | 13·33 | 13 | 1 | 7·69 | 11 | 0 | 0 | 54 | 5 | 9·26 |
| Total général - - | 404 | 13 | 3·21 | 207 | 3 | 1·45 | 158 | 5 | 3·16 | 769 | 21 | 2·73 |

## *Remarques.*

1. Les personnes traitées à St. Pétersbourg ont été réparties en trois catégories de la même manière qu'à l'Institut Pasteur :— 1e catégorie—personnes mordues par des animaux, dont la rage a été reconnue expérimentalement ; 2e catégorie—personnes mordues par des animaux, dont la rage a été reconnue par observations vétérinaires ; 3e catégorie—personnes mordues par des animaux suspects de rage.

2. Dans le compte de la mortalité on fait entrer toutes les personnes prises de rage, même celles chez qui la maladie s'est déclarée pendant le traitement. Deux personnes ont été prises de rage pendant le traitement, et trois autres moins de 15 jours après le dernier jour du traitement.

3. 675 personnes avaient été mordues par des chiens (17 sont mortes) ; 69 par des chats (1 est morte) ; 12 par des loups (3 sont mortes) ; 4 par des chevaux, 3 par des vaches, et 6 par des renards.

# Résultats statistiques de l'Institut Antirabique de Tiflis.

PAR

le Dr. T. Finkelstein, Tiflis.

[This Paper was originally contributed to Section II.]

La station antirabique à Tiflis existe depuis le 1er Juillet 1888; elle forme une section du Laboratoire Médical Militaire du Caucase :—

Du 1er Juillet 1888 au 31 Décembre 1888 nous avons eu 31 mordus.

Pendant 1889 nous avons eu 88 mordus avec 3 morts.

„ 1890 „ „ 94 „ „ 5 „

Du 1er Janvier 1891 au 1er Juillet 1891 nous avons eu 75 mordus avec 1 mort.

Nous commençons le traitement par le moëlle de 14 ou de 12 jours. Nos trépanations nous ont montré que ces moëlles sont souvent virulentes; et nous finissons chacune de deux séries par une moëlle d'un jour; nous faisons en somme 30–35 inoculations pendant 18–20 jours, 2 inoculations par jour. Dans les cas plus graves, chez les mordus par des loups, le nombre des inoculations monte jusqu'à 42–45 (3–5 par jour).

L'âge de nos mordus était :—

| — | 1888. | 1889. | 1890. | 1891. |
|---|---|---|---|---|
| 6½ mois - - - - | — | — | 1 | — |
| Jusqu'à 5 ans - - - | 2 | 9 | 2 | 5 |
| 6–12 ans - - - | 5 | 22 | 12 | 12 |
| 13–18 „ - - - | 2 | 9 | 12 | 9 |
| 19–45 „ - - - | 19 | 42 | 58 | 45 |
| 46–62 „ - - - | 3 | 6 | 9 | 4 |
| Hommes - - - | 27 | 72 | 76 | 69 |
| Femmes - - - | 4 | 16 | 18 | 6 |

Les personnes traitées avaient été mordues :—

| — | 1888. | 1889. | 1890. | 1891. | Total général. | |
|---|---|---|---|---|---|---|
| En Janvier - - | — | 2 | 5 | 10 | 17 | En hiver, 60. |
| „ Février - - | — | 4 | 7 | 16 | 27 | |
| „ Mars - - - | — | 11 | 10 | 7 | 28 | Pendant le printemps, 84. |
| „ Avril - - | — | 16 | 7 | 7 | 30 | |
| „ Mai - - - | — | 4 | 7 | 15 | 26 | |
| „ Juin - - - | 6 | 6 | 16 | 18 | 46 | Pendant l'été, 87. |
| „ Juillet - - - | 7 | 11 | 11 | — | 29 | |
| „ Août - - | 8 | 7 | 2 | — | 12 | |
| „ Septembre - - | 4 | 10 | 9 | — | 23 | Pendant l'automne, 57. |
| „ Octobre - - | 5 | 5 | 9 | — | 19 | |
| „ Novembre - - | 4 | 5 | 6 | — | 15 | |
| „ Décembre - - | 4 | 7 | 5 | — | 16 | |

Nos mordus sont arrivés à la station après les morsures :

| — | 1888. | 1889. | 1890. | 1891. | Total. |
|---|---|---|---|---|---|
| 0–2 jours - - - | 2 | 22 | 20 | 16 | } 164 personnes. |
| 3–7 „ - - - | 8 | 27 | 29 | 33 | |
| Pendant la 2me semaine - | 4 | 22 | 33 | 15 | 74 „ |
| „ 3me „ - | 4 | 6 | 7 | 6 | 23 „ |
| „ 4me „ - | 6 | 1 | 4 | 2 | 13 „ |
| „ 5me „ - | — | 3 | 1 | — | 4 „ |
| „ 6me „ - | — | 2 | — | 2 | 4 „ |
| „ 7me „ - | — | 1 | — | 1 | 2 „ |
| Après 4 mois - - | — | 4 | — | — | 4 „ |

Pendant 1889 sont morts de la rage trois personnes, mordues par des loups enragés :

1. Tchkheïdsé, 50 ans, paysan, mordu le 27 Juillet à la joue gauche, avec fracture des os (maxillaire super. et zygoma). Pas de cautérisation. Il a été mis en traitement le 29 Juillet, et a reçu 28 inoculations pendant 12 jours, jusqu'au 9 Août, quand il est tombé malade. Sa température était 38·8° C., hydrophobie, aërophobie, aucune paralysie la mort survient le 11 Août à l'hôpital de la ville. Le bulbe de Tch. est inoculé à deux lapins, qui sont devenus enragés 14–16 jours après la trápanation et morts après 17–19 jours.

2. David Tsintsadsé, 62 ans, paysan, mordu par le même loup à la jambe droite, où était une grande et profonde blessure. Mis en traitement le 29 Juillet, il a reçu 47 inoculations pendant 30 jours ; il devient malade le 3 Septembre (7 jours après la fin du traitement), rage convulsive ; il est mort le 7 Septembre. Trois autres hommes, mordus par le même loup et traités à la station, sont bien portants jusqu'à présent.

3. Kosanachwili Bans, mordu par un loup le 10 Septembre. Le traitement fut commencé le 20 Septembre. La lèvre supérieure est mordue au travers. K. a reçu seulement 20 inoculations pendant 10 jours ; il est devenu malade le 30 Septembre, T.° 38·3–39·2. Rage convulsive, nulle paralysie. Il mourut à l'hôpital de la ville le 2 Octobre. Le bulbe de K., inoculé aux lapins, provoque chez eux la rage après 16–17 jours.

Pendant 1890 sont morts 5 hommes : 3 mordus par des loups, 1 mordu par un chacal, et 1 mordu par un chien.

1. Paul Khoinia, 12 ans, mordu par un chacal le 4 Mars à la tête. Pas de cautérisation. En traitement du 16 Mars, devient malade le 29 Mars (il n'a reçu que 26 inoculations), T.° 38·3–38·9, il est mort de la rage furieuse le 31 Mars à l'hôpital de la ville. Aucune paralysie. Les cobayes, inoculés avec le bulbe de Kh., succombent à la rage après 16–17 jours. Le bulbe du chacal-mordeur, inoculé aux cobayes, provoque chez eux la mort après 18 jours.

2. Kikela Chwili, 11 ans, mordu par un chien le 30 juin à la tête, au visage et aux mains, 6 morsures, pas de cautérisation. En traitement

du 11 au 29 Juillet (36 inoculations). Rage convulsive le 8 Août (10 jours après le traitement), il est mort le 11 Août. Un autre garçon, mordu par le même chien et traité à notre station, est en bonne santé.

3. Ter-Martirossoff, 37 ans, mordu par un loup le 11 Juillet à la tête. Pas de cautérisation. En traitement du 24 Juillet au 14 Août (42 inoculations). Rage convulsive du 29 au 31 Août, quand il mourut.

4. Ali Ramazan Ogly, 14 ans, mordu le 29 Août par un loup à la joue droite. Pas de cautérisation. En traitement du 10 au 28 Août (45 inoculations). Rage furieuse le 4 Septembre, mort le 6 Septembre.

5. Ramazan Kourban Ogly, 40 ans, mordu par le même loup qu'Ali Ramazan. Cinq grandes et profondes blessures à la main gauche. Pas de cautérisation. Rage convulsive le 21 Septembre, mort le 23 Septembre. Quatre autres personnes mordues par le même loup et inoculées à la station, se portent bien.

Pendant 1891 est mort de la rage Magoma Rakhman Ogly, 20 ans, mordu le 2 Février par un loup à la tête, devient malade 34 jours après les morsures. Son bulbe, inoculé aux animaux, les rend enragés après 22 jours. Un autre homme, mordu par le même loup et inoculé à la station, est en bonne santé.

Ainsi de 9 personnes mortes de la rage, 7 avaient été mordues par des loups, 1 par un chacal, 1 par un chien. Les morsures se trouvaient chez 7 à la tête et au visage, chez 1 à la main, et chez 1 à la jambe. Chez toutes, excepté la dernière, les blessures étaient nombreuses; chez toutes elles étaient graves. De ces 9 personnes sont devenues malades :—

4, après 13, 18, 25, et 34 jours après les morsures et jusque à la fin du traitement.

3, après 37, 38, 38 jours après les morsures et 7–10 jours après le traitement.

1, après 39 jours après les morsures et 15 jours après le traitement

1, après 54 jours „ „ 24 „ „

La mortalité a été de 3·1°/₀. Pour les mordus par des loups la proportion est 24·8°/₀. Pour les mordus par des chiens (sur 245 personnes) la mortalité, 0·408. Si nous retranchons des 9 personnes 8 morts avant les 15 jours qui se sont passés depuis la fin du traitement, nous aurons une mortalité de 0·34°/₀.

---

## Statisque du Traitement préventif de la Rage à la Station de Tiflis (Caucase) du 1/13 juillet 1888 au 1/13 juillet 1891.

Directeur, Dr. T. Finkelstein.

| — | A. | | | | | | | | B. | | | | | | | | C. | | | | | | | | Totaux. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1888.* | | 1889. | | 1890. | | 1891.† | | 1888. | | 1889. | | 1890. | | 1891. | | 1898. | | 1889. | | 1890. | | 1891. | | |
| Morsures à la tête et à la figures - - simples - | . | . } 1 | . | 4 } 6 | . | 4 } 9 | . | . } 5 | . | . } 1 | . | . | . | 1 } 1 | . | . } 2 | . | . | . | 3 } 9 | . | . } 5 | . | . } 6 | 45 |
| Morsures à la tête et à la figures - - multiples | . | 1 | . | 2 | . | 5 | . | 5 | . | 1 | . | . | . | . | . | 2 | . | . | . | 6 | . | 5 | . | 6 | |
| Cautérisation efficace - - | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . |
| „ inefficace - - | 1 | . | 1 | . | . | . | 2 | . | . | . | . | . | . | . | 1 | . | . | . | 4 | . | . | . | 1 | . | . |
| Pas de cautérisation - - | . | . | 5 | . | 9 | . | 3 | . | 1 | . | . | . | 1 | . | 1 | . | . | | 5 | . | 5 | . | 5 | . | . |
| Morsures aux mains - simples - | . | 2 } 2 | . | 10 } 14 | . | 7 } 7 | . | 2 } 6 | . | 2 } 3 | . | 3 } 6 | . | 3 } 7 | . | 3 } 5 | . | } 5 | . | 4 } 15 | . | 7 } 18 | . | 5 } 18 | 101 |
| Morsures aux mains - multiples | . | . | . | 4 | . | . | . | 4 | . | 1 | . | 3 | . | 4 | . | 2 | . | | . | 11 | . | 11 | . | 8 | |
| Cautérisation efficace - - | . | . | 1 | . | . | . | 1 | . | 1 | . | 1 | . | . | . | . | . | 1 | | 1 | . | 2 | . | 2 | . | . |
| „ inefficace - - | . | . | 3 | . | . | . | 1 | . | 1 | . | 3 | . | 5 | . | 1 | . | 3 | | 3 | . | 6 | . | 2 | . | . |
| Pas de cautérisation - - | 2 | . | 10 | . | 7 | . | 4 | . | 1 | . | 2 | . | 2 | . | 4 | . | 1 | | 11 | . | 10 | . | 8 | . | . |
| Morsures aux membres et au tronc - simples - | . | 1 } 4 | . | 4 } 15 | . | 5 } 7 | . | 3 } 6 | . | 1 } 5 | . | 4 } 6 | . | 5 } 11 | . | 3 } 6 | . | 4 } 10 | . | 7 } 17 | . | 8 } 29 | . | 12 } 26 | 142 |
| Morsures aux membres et au tronc - multiples | . | 3 | . | 11 | . | 2 | . | 3 | . | 4 | . | 2 | . | 6 | . | 3 | . | 6 | . | 10 | . | 21 | . | 14 | |
| Cautérisation efficace - - | . | . | 2 | . | . | . | . | . | 1 | . | . | . | . | . | . | . | . | . | . | . | 4 | . | . | . | . |
| „ inefficace - - | 2 | . | 1 | . | 1 | . | . | . | 1 | . | 5 | . | 6 | . | 4 | . | 5 | . | 4 | . | 8 | . | 11 | . | . |
| Pas de cautérisation - - | 2 | . | 12 | . | 6 | . | 6 | . | 3 | . | 1 | . | 5 | . | 2 | . | 5 | . | 13 | . | 17 | . | 13 | . | . |
| Morsures à nu - - - | . | . | 2 | . | 2 | . | . | . | . | . | . | . | 1 | . | . | . | . | . | 2 | . | . | . | . | . | . |
| Totaux - - - | . | 7 | . | 35 | . | 23 | . | 17 | . | 9 | . | 12 | . | 19 | . | 13 | . | 15 | . | 41 | . | 52 | . | 45 | 288 |

* Du 1er Juillet au 31 Decembre. † Du 1er Janvier au 1er Juillet.

| — | 1888. | 1889. | 1890. | 1891. |
|---|---|---|---|---|
| Les animaux mordeurs ont été :— | | | | |
| Chiens - - | 29 fois. | 74 fois. | 75 fois. | 67 fois. |
| Loups - - | — | 7 „ | 15 „ | 7 „ |
| Chats - - | 2 fois. | 1 „ | 1 „ | 1 „ |
| Chacal - - | — | 2 „ | 1 „ | — |

| — | 1888. | 1889. | 1890. | 1891. |
|---|---|---|---|---|
| Les animaux mordeurs ont été :— | | | | |
| Bouc - - - | — | 2 fois. | — | — |
| Cobaye (inoculé) - | — | 1 „ | — | — |
| Enfant - - | — | 1 „ | — | — |
| Cheval - - | — | — | 2 fois. | — |

# Les Résultats des Vaccinations Antirabiques obtenus à Varsovie.

PAR

le Dr. O. Bujwid, Warsaw.

[This Paper was originally contributed to Section II.]

Pendant sept mois de l'année 1886 j'ai traité avec le *traitement simple* 107 personnes mordues, c.-à.-d., commençant par la moëlle de 14 jours, finissant par la moëlle de 5 jours. *Un cas de mort.*

Pendant huit mois de 1887 j'ai traité 193 personnes avec le *traitement affaibli*, c.-à-d., commençant par la moëlle de 6 ou 7 jours de dessèchement. *Huit cas de mort.*

Pendant quatre mois de 1887 et toute l'année 1888 j'ai appliqué chez les 380 personnes le *traitement intensif*, c.-à-d., commençant par la moëlle de 12–10 jours et finissant par la moëlle de 3 ou 2 jours. *Un cas de mort.* Quatre personnes mordues par deux loups, dont la rage avait été constatée par la trépanation des lapins, avaient des morsures très graves à la tête et à la figure. Toutes ces personnes sont restées en bonne santé.

En 1889 j'ai appliqué le traitement intensif chez 343 personnes. Quelques unes de ces personnes mordues légèrement, n'ont reçu le traitement intensif que d'une manière partielle; par exemple une seule série de moëlles, finissant par la moëlle de 3 jours. Deux personnes ainsi traitées mordues aux mains ont succombé à la rage malgré ce traitement. En tout j'ai eu 3 cas de mort.

En 1890 j'ai appliqué seulement le traitement intensif à toutes les personnes mordues par les animaux dont la rage avait été constatée par l'expérience, par l'autopsie ou par les symptômes rabiques; 448 personnes ont subi ce traitement, il n'y a eu qu'un cas de mort, malgré un grand nombre de morsures à la figure et aux mains nues.

Voici la statistique detaillée suivant le classement en usage à l'Institut Pasteur à Paris :—

Il faut remarquer, que depuis 3 ans nous faisons un accueil beaucoup plus sévère parmi nos malades qu'auparavant; 25 °/₀ environ ne subit pas le traitement, car nous pouvons considérer leurs morsures comme inoffensives. Quant au diagnostic de la rage des animaux mordeurs, nous ne sommes pas toujours en possession de moëlles pour faire une expérience sur le lapin; néanmoins nous n'avons pas de grandes différences parmi les chiffres des morts des divers groupes; par exemple, en 1887 il y eut 2 cas de mort parmi les personnes du groupe A.; 3 parmi les personnes du groupe B.; et 3 parmi celles du groupe C. En 1889, 2 personnes du groupe B. et 1 du groupe C.; en 1890, 1 personne du groupe B., sont mortes de rage.

| | 1886. | | | | | | | | | 1887. | | | | | | | | | 1888. | | | | | | | | | 1889. | | | | | | | | | 1890. | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | A. | | | B. | | | | | | C. | | | | | | A. | | | | | | B. | | | | | | C. | | | | | | A. | | | | | | B. | | | | | | C. | | |
| Morsures à la tête et à la figure— | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Simples - - | . | . | . | . | 1 | 2 | . | 1 | 3 | . | 1 | 7 | . | 7 | 10 | . | . | . | . | 2 | 4 | . | 11 | 23 | . | . | . | . | 1 | 3 | . | . | . | 9 | 18 | . | 1 | 2 | . | 4 | 7 | . | 15 | 31 |
| Multiples - - | . | . | . | . | 1 | | . | 2 | | . | 6 | | . | 3 | | . | . | . | . | 2 | | . | 9 | | . | 4 | 4 | . | 2 | | . | 4 | 4 | . | 9 | | . | 1 | | . | 3 | | . | 16 | |
| Cautérisations efficaces - | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | 1 | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | . | 1 | . | . |
| „ inefficaces - | . | . | . | 1 | . | . | 1 | . | . | 6 | . | . | 7 | . | . | . | . | . | 2 | . | . | 7 | . | . | 1 | . | . | . | . | . | . | . | 2 | . | . | . | . | . | 4 | . | . | 10 | . | . |
| Pas de cautérisations - | . | . | . | 1 | . | . | 2 | . | . | 1 | . | . | 3 | . | . | . | . | . | 2 | . | . | 12 | . | | 3 | . | . | 3 | . | . | 4 | . | . | 16 | . | . | 2 | . | . | 3 | . | . | 20 | . | . |
| Morsures aux mains— | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Simples - - | . | 6 | 22 | . | 17 | 27 | . | 8 | 21 | . | 16 | 30 | . | 45 | 88 | . | 15 | 24 | . | 20 | 36 | . | 60 | 155 | . | 11 | 18 | . | 25 | 59 | . | 11 | 26 | . | 50 | 114 | . | 7 | 19 | . | 27 | 50 | . | 109 | 212 |
| Multiples - - | . | 16 | | . | 10 | | . | 13 | | . | 14 | | . | 43 | | . | 9 | | . | 16 | | . | 95 | | . | 7 | | . | 37 | | . | 15 | | . | 64 | | . | 12 | | . | 23 | | . | 103 | |
| Cautérisations efficaces - | . | . | . | 1 | . | . | 3 | . | . | 15 | . | . | 5 | . | . | . | . | . | 15 | . | . | 10 | . | . | 3 | . | . | 1 | . | . | . | . | . | 2 | . | . | 1 | . | . | 1 | . | . | 7 | . | . |
| „ inefficaces - | 6 | . | . | 19 | . | . | 8 | . | . | 14 | . | . | 60 | . | . | 16 | . | . | 21 | . | . | 76 | . | . | 4 | . | . | 11 | . | . | 7 | . | . | 22 | . | . | 3 | . | . | 18 | . | . | 64 | . | . |
| Pas de cautérisations - | 16 | . | . | 7 | . | . | 10 | . | . | 1 | . | . | 23 | . | . | 8 | . | . | . | . | . | 69 | . | . | 11 | . | . | 47 | . | . | 19 | . | . | 90 | . | . | 15 | . | . | 31 | . | . | 174 | . | . |
| Morsures aux membres et au tronc— | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Simples - - | . | 1 | 2 | . | 8 | 17 | . | 6 | 10 | . | 14 | 24 | . | 35 | 55 | . | 6 | 17 | . | 5 | 7 | . | 22 | 60 | . | 8 | 13 | . | 18 | 39 | . | 6 | 7 | . | 33 | 73 | . | . | . | . | 1 | 20 | . | 3 | 96 |
| Multiples - - | . | 1 | | . | 9 | | . | 4 | | . | 10 | | . | 20 | | . | 11 | | . | 2 | | . | 38 | | . | 5 | | . | 21 | | . | 1 | | . | 40 | | . | 11 | 11 | . | 19 | | . | 93 | |
| Cautérisations efficaces - | 1 | . | . | 2 | . | . | 2 | . | . | . | . | . | 3 | . | . | . | . | . | . | . | . | 3 | . | . | . | . | . | . | . | . | 1 | . | . | 3 | . | . | . | . | . | . | . | . | 2 | . | . |
| „ inefficaces - | . | . | . | 9 | . | . | 2 | . | . | 10 | . | . | 36 | . | . | 7 | . | . | 2 | . | . | 20 | . | . | 4 | . | . | 6 | . | . | 1 | . | . | 11 | . | . | 2 | . | . | 3 | . | . | 30 | . | . |
| Pas de cautérisations - | 1 | . | . | 6 | . | . | 6 | . | . | 14 | . | . | 16 | . | . | 10 | . | . | 5 | . | . | 37 | . | . | 9 | . | . | 33 | . | . | 5 | . | . | 59 | . | . | 9 | . | . | 17 | . | . | 64 | . | . |
| Habits déchirés - - | . | 2 | 24 | . | 15 | 46 | . | 12 | 34 | . | 36 | 61 | . | 41 | 153 | . | 25 | 41 | . | 9 | 47 | . | 98 | 235 | . | 5 | 35 | . | 24 | 101 | . | 6 | 37 | . | 61 | 205 | . | 14 | 32 | . | 24 | 77 | . | 101 | 339 |
| Morsures à nu - - | . | 22 | | . | 31 | | . | 22 | | . | 25 | | . | 112 | | . | 16 | | . | 38 | | . | 137 | | . | 30 | | . | 77 | | . | 31 | | . | 144 | | . | 18 | | . | 53 | | . | 238 | |
| Totaux - | 104 | | | | | | | | | 255 | | | | | | | | | 317 | | | | | | | | | 343 | | | | | | | | | 448 | | | | | | | | |

## DISCUSSION.

**Dr. T. W. Hime** (Bradford) said that he would not discuss the Pasteur method, because he believed all persons who had studied the question were satisfied that it was established on a scientific basis, beyond all doubt. He would only mention one case within his own knowledge: 2½ years ago a dog of his was inoculated with the rabic virus under the *dura mater*, and was then put through a course of treatment with the same material and in the same manner as used for human beings. That dog was still alive, and had never had a symptom of disease, while everyone of the control animals had died of rabies, not having received the protective inoculations after the inoculation with rabic virus. He thought it should be compulsory that all dogs should wear collars with the address of the owners thereon, because it would be a positive advantage to know where rabid animals came from. This would enable special precautions to be taken in the district whence the disease had emanated, so as to meet the probable danger arising from dogs bitten in the infected area. At present, the difficulty of ascertaining where the infected animal came from, increased the difficulty of suppressing the disease. Moreover, by this means, the owner of the dog could be discovered, and his responsibility brought home to him. Our law should be so altered that the owner of a dog which had bitten a person should be readily open to conviction, just as a person responsible for any other form of personal injury, seemed self-evident. At present it was necessary to prove that the owner knew his dog was vicious before it had bitten anyone, in order to secure conviction. He suggested that a much higher dog-tax would lead to the destruction of thousands of useless and neglected dogs. Muzzling might be useful, if carried out rigorously and generally. The present practice in England of muzzling dogs in one area, while the contiguous ones were left unmuzzled, was manifestly useless and absurd. It was not correct to assume that it was only wandering dogs which required muzzling, or which ever became rabid. The most valuable and apparently best cared for dogs became rabid. Whatever measures were required to deal with the one class, must be so regulated as to be generally applicable to the canine population. He wished to direct attention to some facts not, he believed, generally known, which rendered the necessity of surpervising dogs more pressing. It had been demonstrated by MM. Roux and Nocard that even three days before the slightest symptoms of rabies showed themselves in an infected dog, under careful observation, its saliva was infective, and when inoculated would cause rabies in dogs and other animals. This showed how very important it was to know whence a rabid dog came, so that extra precautions might be taken throughout the area. That rabies could be entirely eradicated was indubitable, and that it was still existing in our midst was characteristic of our apathy. In conclusion, he asked whether there were any facts available as to the question why the dogs of Australia, where rabies was unknown, were less susceptible to rabies than the dogs of other countries; or whether this fortunate immunity of Australian dogs was entirely due to the measures of quarantine of three months carried out there against all imported dogs.

**Professeur Nocard** (Alfort) dit: Je voudrais vous dire quelques mots d'un traitement prophylactique après morsure qui est applicable aux animaux domestiques, à quelques uns d'entre eux au moins, aux herbivores. Notre distingué collègue de Lyons, M. Galtier, a annoncé, il y a déjà

longtemps, que l'injection de virus rabique dans les veines du mouton et de la chèvre ne donne pas la rage, et, qu'au contraire, elle confère l'immunité ; avec M. Roux nous avons montré que les faits annoncés par M. Galtier sont vrais, non seulement de la salive rabique, mais encore de la matière nerveuse qu'on l'emprunte à la *rage des rues* ou à la *rage de passage*. Nous avons pu préserver de la rage des moutons qui avaient été inoculés par injection intra-oculaire depuis déjà 24 heures ; tout récemment, enfin, nous avons fait des expériences encore inédites, prouvant, 1° que l'injection intra-veineuse de virus rabique ne donne pas la rage aux équidés ; 2° que si la quantité de virus injectée a été suffisante, les équidés inoculés ont acquis l'immunité contre la rage ; 3° qu'il est possible de préserver les équidés inoculés par injection intra-oculaire depuis 24 heures déjà, en leur injectant dans les veines, à diverses reprises, de notables quantités de matière nerveuse rabique diluée (de la rage de passage, de préférence).

Si l'on songe au nombre considérable d'herbivores (chevaux, bœufs, moutons) que prennent la rage lorsqu'ils ont été mordus par des chiens enragés (près de 80 °/₀), si l'on envisage la valeur pécuniaire élevée de ces animaux, on juge des services que peut rendre la méthode de traitement prophylactique qui procède des travaux de M. Galtier.

Je désire aussi dire quelques mots sur la question de la prophylaxie de la rage canine. En principe, je suis, cela va sans dire, entièrement de l'avis de notre distingué collègue, le Dr. Fleming. Il est incontestable que partout où la muselière est rigoureusement appliquée, la rage diminue rapidement. Mais il est clair que ce résultat ne tient qu'indirectement à la muselière. Jamais une muselière n'a empêché un chien enragé de mordre ; quand on a vu un chien enragé se briser les dents et les mâchoires sur les barreaux de sa cage, on conçoit que ce chien ne garderait pas une minute la muselière qu'on aurait pu lui mettre avant l'accès. Mais l'obligation de la muselière a cet avantage, de signaler clairement à la police les chiens qui se sont échappés de la maison, ou qui ne sont l'objet d'aucune surveillance. Or, ces sont ceux-ci qui fournissent la grande majorité des chiens enragés ; les saisir et les prendre, c'est supprimer la plupart des candidats à la rage.

On parviendrait au même résultat, en prescrivant que tout chien devra, sur la voie publique, être tenu en laisse ; c'est ce qui s'est produit, à Paris, en 1888. On avait observé, pendant le mois d'avril, 125 chiens enragés ; le préfet de police prend une ordonnance prescrivant la tenue en laisse de tous les chiens. La mesure ne fut appliquée que pendant six semaines, et cependant elle suffit pour réduire de 125 à 25 le nombre mensuel des chiens enragés pour le dernier semestre de 1888.

La vérité est qu'il n'est pas besoin de nouvelles mesures san pour supprimer la rage ; il suffit de vouloir et d'appliquer exactement rigoureusement, les mesures très simples qui sont prescrites actuellemen dans tous les pays civilisés.

**Dr. C. R. Drysdale** (London) having frequently attended the inoculations at the Pasteur Institute, could speak with the very greatest admiration of the immense care and intelligence shown in the carrying out of the treatment. As to the result of Pasteur's plan, he considered it now clearly proved. When only 0·92 per cent. of deaths had occurred after this treatment, as compared with probably more than 20 per cent. in similar cases not treated, it was an extravagant scepticism to deny its efficacy any longer. Consequently, he thought it was high time that a Pasteur institute should be formed in London, for it was not fair for us to

send over all our patients to Paris, and then to abuse Pasteur and his noble-minded assistants. As to the prevention of rabies, England could apply a muzzling law better than any other European country, because it was surrounded by the sea; and if muzzling were uniformly enforced for a year, the disease might be stamped out of the United Kingdom, and when once stamped out, it might, as in Australia and New Zealand, be kept out by means of quarantine. There was no doubt that rabies was an eminently preventible disease, and, being so, it was extremely immoral in any one to refuse to help forward such a salutary law in this or in any other country, as, for instance, the muzzling law of Prussia.

**Professor Redfern** (Belfast) directed attention to the fact that hydrophobia differed from other diseases in being absolutely incurable, and in being attended with horrible sufferings incapable of relief. The Section was evidently unanimous in the belief that hydrophobia might be entirely extinguished as a disease in the British Isles by the methods suggested by Dr. Fleming, and also that, except in very rare cases, persons bitten by mad dogs might by the methods of M. Pasteur be prevented from suffering the agonies of hydrophobia. He thought it very desirable that the Section should consider carefully whether persistent efforts to call the attention of their legislators to the conclusions arrived at would not certainly result in legislation which would put an end to hydrophobia altogether in Great Britain.

**Dr. Ostertag** (Stuttgart) who spoke in German, said it had been objected that the muzzle was ineffective, because cats and wolves carried the disease as well as dogs; but that cases of this sort were rare was proved by the experienc of Germany. Since the passing of laws regulating the contagious diseases of animals, rabies had become a rare disease, and only occurred along the French and Russian borders. These laws provided that all mad dogs, and those suspected of madness, or even of having bitten, must be killed, and all dogs and cats within a radius of 15 miles must be kept in confinement for three months. In Berlin all dogs wore muzzles, and a case of hydrophobia had not been reported for 10 years. As a result of these laws, a Pasteur Institute in Germany was unnecessary.

**Sir Henry Simpson, F.R.C.V.S.,** (Windsor) warned the Section that in any recommendation which might be made to the Legislature, provision must be made for exemptions. He would exempt sporting dogs, because for seven or eight months in the year these were being used for hunting, &c. Hunting and other sports led to the employment of many persons, and to the consequent expenditure of money. If men of wealth were asked to sacrifice their hunting for a couple of years they would not do it, and the country would not sanction a measure which would result in such a loss of revenue to the trade of the country. Considering that sporting dogs were well cared for, that their owners were true lovers of dogs, it appeared to him that they need not be muzzled. It should be a second condition that they should be kept in charge of competent persons, that any deviation from health should be at once noted, and an inspection made by a properly qualified veterinary surgeon, and, if necessary, the animals kept in quarantine.

**Dr. Elizabeth Blackwell** (London) called attention to the sufferings of dogs inoculated experimentally. She had visited the Pasteur Institute, and had herself seen a series of dogs in the various stages of induced rabies.

**Dr. Roux,** in reply to Dr. Elizabeth Blackwell, denied emphatically that the virus taken from the dog was, or ever had been, used for inoculating other dogs in order to provide the protective material. The only virus thus used was that obtained from rabbits.

**Dr. Hime,** in drawing attention to this denial, said they now had it upon the highest authority, viz., from Dr. Roux, as representing the head of the Institute, that it was untrue, as had been erroneously stated throughout the country, that dogs by the thousand were made mad at the Pasteur Institute for the purpose of providing the anti-rabic virus.

**Professor Brown, C.B.** (London) said that, as the head of the department which was charged with the duty of checking the progress of contagious diseases of animals, he wished to refer to some of the questions which had been dealt with during the discussion. He agreed with every word which Dr. Roux had uttered as to the value of M. Pasteur's method, and with all that Dr. Fleming had urged in favour of preventive measures; but he wished to emphasise the difficulties which arose from the sentimental objections of owners of animals, who, as Dr. Roux had remarked, were quite incapable of realising that their own dogs might become mad, although they were keenly alive to the risk incurred by their neighbour's dogs, and believed in the necessity of imposing restrictions on them. He could assure the Section that if they, by a strong expression of opinion, could induce the Legislature to insist that all the dogs in the United Kingdom should be muzzled for a certain time, everyone in his Department would be gratified. But he need hardly remark, that an Order to that effect, emanating from the Board of Agriculture, even if they had power to pass one, would excite unusual opposition.

Before concluding, he desired, at the request of Professor Perroncito, who was not able to be present at that particular discussion, to state that the municipality of Turin had a Pasteur laboratory for anti-rabic vaccination, which gave very satisfactory results, as shown by the statistics published by the Director. The same process had also been tried on other animals, such as horses and oxen, with encouraging results.

## Trichinosis in the Netherlands.

BY

Dr. B. Carsten, The Hague.

The first trace of trichinosis in the Netherlands was discovered in 1883 at Jerseke, a village near Goes, in South Beveland, one of the islands forming the province of Zeeland, whence, by means of the pigs kept at, and the rats infesting, a tannery and storage for offal, the disease spread till 140 persons in seven different parishes on the island were affected, one succumbing.

The disease, which threatened to become enzootic in this district, was stamped out, thanks to the energetic measures adopted: — A Royal decree was issued prohibiting the removal of pigs and of pig manure from an infected district, and a special veterinary inspector was stationed temporarily in the neighbourhood as supervisor; the local authorities instituted a strict research for trichinæ, ordering that all pork before consumption be submitted for inspection to a competent committee appointed for that purpose, that all pig-yards be overhauled, and that wherever any trace of trichinæ be found the infected swine and all vermin found in the neighbourhood thereof be killed and, together with the offal, consumed by fire.

Except that at South Beveland, the only other appearances of trichinosis in the Netherlands occurred in 1888, at Leyden and Noordwijk, two places in close proximity in the province of South Holland, where prompt action stopped its spread. Since 1889 there has been no return of the disease in the Netherlands.

In March 1888, trichinosis was included among infectious cattle diseases, and consequently it was brought under the law, thus making all infected pork liable to seizure and destruction by order of the local veterinary official.

That trichinosis is now a thing of the past in the Netherlands is due to the thorough organisation and the persistent enforcement of precautionary measures.

## Les Parasites transmissibles des Animaux a l'Homme, envisagés spécialement au point de vue de la Prophylaxie.

PAR

le Professeur Railliet, Alfort.

On peut dire que, dès les temps les plus anciens, la croyance était répandue que l'homme contracte bon nombre de ses parasites au contact des animaux. C'était là, sans doute, une opinion basée en grande partie sur des observations inexactes, et nous la voyons encore fréquemment aujourd'hui s'affirmer dans le peuple en raison d'une simple ressemblance grossière et lointaine entre les parasites de l'homme et ceux des animaux. Mais il est vraisemblable, cependant, que les anciens observateurs avaient reconnu, d'une façon plus ou moins précise, a transmission réelle de certains parasites des animaux à l'homme. Pour n'en citer qu'un exemple, nous rappellerons la prohibition de la viande de porc par les prêtres égyptiens du temps des Pharaos et par Moïse, attribuable sans doute à la transmission du *Tænia solium* et

peut-être même de la Trichine; du moins est-on en droit de supposer que l'usage de cette viande avait été reconnu comme susceptible de provoquer des troubles graves dans la santé de l'homme.

Mais les données relatives à la transmission des parasites ne sont entrées dans une phase réellement scientifique que dans le courant du siècle actuel, soit plus exactement à l'époque où ont été établies, d'une part, la nature parasitaire des teignes et des gales, d'autre part, les migrations des Ténias. Depuis lors, en effet, cette transmission a été mise hors de doute, dans un grand nombre de cas, soit par des observations précises et répétées, soit surtout par des recherches expérimentales.

Nous nous proposons de passer rapidement en revue, dans ce travail, les parasites des animaux transmissibles à l'homme, en nous limitant d'ailleurs aux zooparasites ou parasites animaux, les phytoparasites devant être envisagés spécialement par M. le Dr. Blanchard.

La série que nous aurons à examiner est du reste déjà très étendue. L'homme, en sa qualité d'omnivore, pouvant donner asile à la fois aux parasites des herbivores et à ceux des carnivores. Aussi devrons-nous nous borner à une simple esquisse de l'histoire de chacun des types signalés, en insistant de préférence, chaque fois que la chose sera possible, sur leur évolution, de façon à dégager, de la connaissance de leur mode de propagation, les moyens propres à les éviter.

Il est presque inutile de faire remarquer que la très grande majorité des parasites dont nous aurons à nous occuper sont fournis à l'homme par les animaux domestiques. Le fait s'explique de lui-même par la fréquence des rapports que nous entretenons avec ces animaux, et du rôle qu'ils jouent dans notre alimentation.

Sans nous étendre plus longuement sur ces remarques générales, nous classerons immédiatement les parasites, d'après leur siège, en deux grandes sections :—parasites externes ou *ectoparasites*, et parasites internes ou *endoparasites*.

I. Parasites externes.—Les ectoparasites peuvent se grouper, d'une façon assez nette, d'après le degré de leur parasitisme. Les uns, en effet, sont des parasites *libres* ou *temporaires*, c'est-à-dire ne vivant sur leur hôte qu'au moment même où ils viennent se nourrir à ses dépens, et le quittant aussitôt, ou tout au moins étant susceptibles de le quitter pour vivre en liberté. Les autres, qualifiés de *stationnaires*, restent à demeure chez cet hôte dès l'instant où ils ont fixé chez lui leur séjour.

*Parasites externes temporaires.*

| | |
|---|---|
| Tabanidæ. | Pulex. |
| Stomoxys. | —— |
| Glossina. | Dermannyssus. |
| Hippobosca. | Argas. |
| Simulium. | |

A.—Les parasites *temporaires* n'offrent, au point de vue où nous devons nous placer ici, qu'un intérêt secondaire. Vivant à peu près

constamment en liberté, ils se jettent, dès qu'ils sont poussés par la faim, sur les animaux qui se trouvent à leur portée, dans le but de se repaître de leur sang. C'est donc à peine s'ils méritent la qualification de parasites; et comme d'ailleurs ils attaquent l'homme presque au même titre que les animaux; que, par suite, ils ne sont pas à proprement parler transmis de ceux-ci à celui-là, nous nous bornerons à les mentionner.

Ce sont des Insectes et des Acariens.

Parmi les premiers se rangent les Taons (*Tabanidæ*), dont les femelles, avides de sang, se jettent d'ordinaire sur les chevaux et les bœufs, mais piquent également l'homme à l'occasion. A côté d'eux, il faut citer d'autres Diptères, de la famille des *Muscidæ*, tels que le Stomoxe mutin (*Stomoxys calcitrans* Geof.), qui harcèle particulièrement les chevaux, mais qui ne laisse pas cependant d'attaquer aussi l'homme, et que l'on peut considérer comme un porte-virus, susceptible d'inoculer spécialement le charbon lorsqu'il a souillé sa trompe au contact d'un animal charbonneux ou même d'un cadavre; puis la fameuse Tsétsé (*Glossina morsitans* Westwood), de la zone torride africaine, dont le rôle paraît être à peu prés le même, mais qui, en tout cas, ne doit pas inoculer un venin propre, ainsi qu'il résulte d'une expérience de Nocard et Railliet.

Dans la famille des *Hippoboscidæ*, nous avons à signaler l'Hippobosque du cheval (*Hippobosca equina* L.), qui attaque la plupart de nos animaux domestiques, mais n'est pas beaucoup moins avide du sang de l'homme, comme en témoignent une expérience de Réaumur et une observation de Paullini. Citons encore, dans une famille voisine, les Simulies (*Simulium*), auxquelles se rattachent la plupart des Diptères connus sous les noms de *Moustiques* ou *Mosquitos*, et qui souvent se jettent, en nombre immense, sur les animaux et sur l'homme; on peut admettre que leur trompe pénétrante est apte à inoculer les virus dont elle a pu se souiller. Enfin, c'est parmi les parasites temporaires qu'il convient aussi de placer les Puces (*Pulex*), puisque ces Insectes sont susceptibles de vivre un temps plus ou moins long en dehors de leur hôte. Les Puces qui vivent sur l'homme et sur les animaux appartiennent à des espèces distinctes et assez nombreuses; or, il y a lieu de se demander si les Puces des animaux sont susceptibles de piquer l'homme. Pour mon compte, j'ai longtemps répondu à cette question par la négative; bien souvent on m'avait communiqué, comme étant certainement des Puces de chien, des individus pris en flagrant délit de morsure sur l'homme, et j'avais toujours constaté qu'il s'agissait de la véritable Puce de l'homme. Pourtant, il est bien avéré que la puce du chien (Dugès), comme celle de la poule (Lucet, Railliet), peut piquer l'homme; mais le fait est beaucoup plus rare qu'on ne le pense habituellement.

Quant aux Acariens qui se classent parmi les parasites temporaires, ils ne comprennent guère que les Dermanysses et les Argas.

Le Dermanysse des volailles (*Dermanyssus gallinæ* Degeer) est un animal noctambule; il se tient pendant le jour dans les fissures des

poulaillers et des colombiers, et, la nuit venue, se jette sur les oiseaux, dont il suce le sang. Dans des cas exceptionnels cependant, son parasitisme devient stationnaire, et il vit à demeure, en quantité extraordinaire, sur les pigeons, sur les poules, et surtout sur les poussins. D'autre part, lorsque ces Acariens sont très abondants, ils ne bornent pas leurs attaques aux oiseaux, mais se jettent volontiers sur les mammifères qui se trouvent à leur portée. C'est ainsi que l'homme est exposé à leurs atteintes. Alt, Bory de Saint-Vincent, Raspail, Simon, etc., ont cité des exemples de transmission de ces parasites à l'homme, et l'on peut ajouter, avec Besnier et Doyon, qu'il n'est pas rare d'observer, chez les garçons ou filles de basse-cour, ainsi que chez les personnes employées à manier et à plumer les volailles, une éruption prurigineuse reconnaissant une telle origine, éruption qui ressemble à certaines formes de l'eczéma papuleux de la gale ordinaire, et qui siège sur le dos des mains, sur l'avant-bras, plus rarement sur la généralité du tronc. Cette affection est nécessairement passagère, car les Dermanysses ne s'acclimatent pas sur la peau de l'homme. La transmission ne pouvant être évitée dans la plupart des cas, on ne peut que recommander de calmer le prurit au moyen de lotions ou de bains d'eau pure ou amidonnée.

On a signalé aussi le passage sur l'homme de quelques autres espèces de Dermanysses, telles que *Dermanyssus avium*, qui vit dans les cages des petits oiseaux, et *Dermanyssus hirundinis*, qui habite les nids d'hirondelle. Les troubles occasionnés sont de même ordre que ceux signalés à propos de l'espèce précédente.

Les Argas, comme les Dermanysses, sont des suceurs de sang. L'Argas bordé (*Argas marginatus* Fabr.) est assez commun en France et en Italie; par contre, il paraît rare en Allemagne et en Angleterre. Il vit dans les colombiers et se répand en plus ou moins grand nombre sur les pigeons. J'ai remarqué que les larves sont souvent fixées à demeure sur le corps de ces oiseaux; mais les adultes ne paraissent les attaquer qu'à de certains moments, la nuit en particulier, et on les découvre d'ordinaire, pendant le jour, cachés dans les fissures des boiseries, les crevasses des murs, etc. Ils se propagent facilement d'un local à l'autre, à la faveur des moindres solutions de continuité; c'est ainsi qu'ils pénètrent quelquefois dans les habitations, et arrivent à se jeter sur l'homme. Raspail, Boschulte et Chatelin en ont cité des exemples. Ce dernier observateur a constaté sur un enfant des piqûres douloureuses et un œdème assez persistant, dus à des Argas d'un colombier évacué depuis plusieurs années. Les Acariens avaient envahi le premier étage et le rez-de-chaussée de l'habitation dans laquelle était situé le colombier. Diverses personnes avaient été atteintes en même temps que cet enfant. Aucune ne présenta de symptômes généraux. Cette espèce peut vivre fort longtemps sans prendre de nourriture; j'en ai vu survivre à 14 mois de jeûne.

Il convient de citer en outre ici *Argas Tholozani*, qui vit en Perse, où il s'attaque aux moutons; *Argas turicata*, du Mexique, qui vit sur le porc; *Argas Megnini*, également du Mexique, qui se rencontre sur

le cheval, l'âne et le bœuf. Toutes ces espèces, et d'autres encore, sucent plus ou moins volontiers le sang de l'homme.

Relativement à ces divers parasites, la prophylaxie doit reposer avant tout, comme en ce qui concerne les Dermanysses, dans la désinfection des locaux où ils abondent.

*Parasites externes stationnaires.*

A.—*Périodiques.*

| | |
|---|---|
| Ochromyia. | Sarcopsylla. |
| Sarcophaga magnif. | —— |
| Hypoderma. | Ixodes ricinus, etc. |
| Dermatobia. | Trombidion (Rouget). |

B.—*Permanents.*

| | |
|---|---|
| Sarcoptes scabiei— | Sarcoptes minor— |
| (Cheval.) | (Chat.) |
| (Bœuf ?) | (Lapin ?) |
| (Mouton.) | |
| (Chèvre.) | Sarcoptes mutans ? |
| (Dromadaire.) | Psoroptes. Chorioptes. |
| (Lama.) | Demodex folliculorum. |
| (Porc.) (Sanglier.) | |
| (Chien.) | |
| (Lion.) (Loup ?) (Renard.) | |
| (Wombat.) | |

B.—Parmi les parasites *stationnaires*, il en est encore qui vivent en liberté pendant une certaine période de leur existence, c'est-à-dire dont le parasitisme n'est que *périodique*, tandis que les autres sont soumis à la condition parasitique depuis leur éclosion jusqu'à leur mort, offrant ainsi un parasitisme *permanent*.

*a*. Comme parasites périodiques, nous avons encore à signaler des Insectes Diptères, appartenant aux familles des *Muscidæ*, des *Œstridæ* et des *Pulicidæ*, et quelques Acariens.

Aux *Muscidæ* appartiennent les *Ochromyia* et les *Sarcophaga*.

L'Ochromyia anthropophage (*Ochromyia anthropophaga*, Blanch.) est plus connue sous le nom de *Mouche du Cayor*. Elle vit dans certaines régions du Sénégal, et passe pour déposer ses œufs dans le sable. Sa larve, dite *Ver du Cayor*, pénètrerait de là dans la peau des animaux et de l'homme lui-même, où elle se développe dans l'espace d'un septénaire.

La Sarcophage magnifique (*Sarcophaga magnifica*, Schiner), répandue dans toute l'Europe, notamment en Russie, dépose ses larves dans les plaies ou dans les cavités naturelles de l'homme ou des animaux. Il en est probablement de même de divers autres Muscidés.

*Les Œstridæ* nous offrent à considérer les Hypodermes et des Dermatobies.

La larve de l'Hypoderme du bœuf (*Hypoderma bovis*, Latr.) se développe d'ordinaire, comme on le sait, dans le tissu conjonctif sous-cutané des bêtes bovines ; plus rarement on l'observe chez le cheval, où elle n'arrive pas à son complet développement ; enfin on possède un certain nombre d'observations relatives à sa présence dans la peau de l'homme. On cite également des cas de développement, sur l'homme, de *l'Hypoderma Diana*, Br., et il est probable que d'autres espèces encore peuvent offrir des exemples semblables.

La Dermatobie funeste (*Dermatobia noxialis*, J. Goudot), de l'Amérique tropicale, se développe à l'état larvaire sous la peau des bœufs et des chiens, et il n'est pas rare de la voir également envahir la peau de l'homme.

Enfin, les *Pulicidæ* comprennent aussi un parasite périodique, la Puce pénétrante ou Chique (*Sarcopsylla penetrans*, L.), originaire de l'Amérique tropicale et récemment introduite en Afrique. Cette Puce s'attaque à la plupart des mammifères et des oiseaux, ainsi qu'à l'homme lui même ; et si le mâle et la femelle jeune sont de simples parasites libres ou temporaires, par contre la femelle fécondée s'introduit à demeure dans la peau.

Les Acariens qui doivent être comptés comme parasites *stationnaires périodiques* ne comprennent que les Ixodes et les Trombidions.

L'Ixode ricin (*Ixodes ricinus*, L.), est particulièrement commun sur les chiens de chasse, mais on peut le rencontrer aussi sur d'autres animaux, tels qui les moutons et les bœufs. De plus, on l'observe de temps en temps sur l'homme, en particulier chez les chasseurs ou chez les individus qui parcourent les landes et les fourrés. Les larves et les nymphes se montrent souvent en abondance sur le corps des petits mammifères, tels que lièvres, lapins, furets, etc. ; mais la femelle fécondée seule se fixe à demeure sur la peau, dans laquelle elle enfonce son rostre pour absorber le sang. Le plus souvent, cependant, sa présence ne détermine aucun accident, comme l'ont montré les observations de Dubreuilh, Moquin-Tandon, J. Chatin, etc. J'en possède moi-même un exemplaire recueilli sur le bras d'une dame qui n'avait éprouvé qu'une certaine douleur et quelques démangeaisons. Par exception, la piqûre de cet Acarien est le point de départ d'accidents fort graves, comme ceux qu'a signalés Raymondand, en 1884, au Congrès médical de Copenhague, accidents qui pourraient même entraîner la mort, d'après Chillida.

D'autres espèces du même genre sont sans doute susceptibles de s'attaquer à l'homme ; mais en somme, comme dans tous les cas précédents, il ne s'agit pas réellement d'une transmission par l'intermédiaire d'un animal.

Ce sont les Trombidions qui vont nous fournir le premier exemple d'une telle transmission. Le Lepte automnal ou Rouget des auteurs est en effet considéré comme la larve hexapode du Trombidion soyeux (*Trombidium holosericeum*, L.). On le rencontre principalement sur les petits mammifères : taupes, lièvres, lapins, etc. ; mais il n'est pas très rare non plus sur les chiens, les chats, et même les oiseaux.

D'autre part l'homme est fréquemment envahi lorsqu' il fréquente, en automne, les taillis ou les plants de groseillers, haricots, etc. Latreille, Raspail, Jahn, Moses, Gudden, White, Johnston, Murray, etc., ont signalé les éruptions et les démangeaisons insupportables occasionnées par ces petits Acariens. Mais, ce qu'il importe de signaler ici, c'est que ces parasites sont susceptibles de se transmettre directement de l'animal à l'homme; c'est ainsi que le regretté T. S. Cobbold a contracté une éruption sur le bras, en caressant un lapin de garenne qui était couvert de Rougets.

*β.*—Nous arrivons enfin aux parasites stationnaires *permanents*, qui tous vont nous offrir des exemples de transmission directe des animaux à l'homme.

Il s'agira exclusivement d'Acariens appartenant aux familles des *Sarcoptidæ* et des *Demodicidæ*, Acariens qualifiés de psoriques en raison des lésions (psore, gale) qu'ils déterminent dans le tissu cutané.

Les Sarcoptidés psoriques ou *Sarcoptinæ* comprennent les trois genres *Sarcoptes*, *Psoroptes* et *Chorioptes*, dont le premier seul présente à notre point de vue un réel intérêt, précisément par ce fait que seul il comprend des formes transmissibles à l'homme.

Le Sarcopte de la gale (*Sarcoptes scabiei*, Latr.) est une espèce bien connue et dont nous n'avons nullement à refaire ici l'histoire. Nous rapellerons seulement qu'il y a lieu d'en distinguer plusieurs variétés, caractérisées principalement par leurs dimensions et par quelques autres particularités morphologiques. Une de ces variétés a pour hôte spécial l'homme ; un grand nombre d'animaux, domestiques ou sauvages, possèdent également leur variété propre; mais il est aujourd'hui bien établi, de par l'observation et l'expérience, que ces diverses variétés peuvent passer d'une espèce animale sur l'autre, ainsi que sur l'homme lui-même, et que, réciproquement que la variété de l'homme peut être communiquée aux animaux.

Nous allons examiner rapidement celles de ces variétés dont la transmission à l'homme a été nettement constatée.

*Gale sarcoptique du cheval.*—Cette forme de gale, dont l'Acarien (*Sarcoptes scabiei*, var. *equi*) a été découvert en 1846 par Eichstedt, est de toute évidence celle dont les anciens auteurs avaient reconnu la transmissibilité à l'homme bien avant que la distinction fût établie entre les trois formes de psore dont le cheval peut être affecté. Les premiers cas de cette contagion du cheval à l'homme paraissent avoir été signalés par Enaux et Chaussier en 1785 ; mais, depuis lors, on en a rapporté un nombre tel que leur simple analyse suffirait à remplir un mémoire spécial. Nous ne pouvons donc qu'indiquer sommairement les plus saillants. Robert Fauvet, vétérinaire italien, rapporte qu'en 1820, un fermier avait acheté au marché de Bergame un cheval galeux qu'il monta pour se rendre chez lui: le lendemain il éprouva une forte démangeaison sur tout le corps. Le palefrenier à qui on confia le cheval se gratta beaucoup le lendemain du pansement fait à cet animal. Ces deux hommes communiquèrent ensuite la gale à d'autres personnes de la ferme, et successivement plus de 30 personnes furent atteintes de la

même maladie. Le cheval galeux ayant été alors vendu à un meunier, celui-ci ne tarda pas à être envahi lui-même, ainsi que ses garçons, qui avaient touché l'animal. Le caractère psorique de l'affection fut reconnu par des médecins distingués. Mais les exemples les plus caractéristiques de contagion s'observent dans les Écoles vétérinaires et dans les régiments de cavalerie. C'est ainsi que Delafond, en 1856, a vu plusieurs élèves de l'École d'Alfort contracter la gale dans une seánce d'exercices de chirurgie portant sur un cheval galeux. De même, Sik raconte qu'une gale qui sévissait, en 1791, dans un régiment de hussards anglais, se communiqua à plus de deux cents cavaliers. Gerlach a fait du reste, sur lui-même et sur des élèves de l'École vétérinaire de Berlin, des essais de transmission du Sarcopte du cheval ; il a vu ainsi la gale se développer à des degrés divers. Chez quelques-uns de ces élèves, elle a été limitée à une période de trois semaines au plus, et s'est alors guérie spontanément ; sur d'autres, elle a persisté de dix-neuf à trente jours, et a nécessité, pour être guérie, des lotions alcalines, suivies d'ablutions faites avec l'eau phagédénique.

En somme, les faits d'observation, ainsi que ceux d'ordre expérimental, démontrent que la gale sarcoptique du cheval se transmet à l'homme ; mais qu'en général elle est assez fugace et tend à disparaître spontanément. Aussi bien, cette transmission est-elle relativement rare, si l'on considère la fréquence de la maladie chez le cheval.

Le bœuf ne paraît pas posséder de Sarcopte spécial, et les faits de transmission de la gale du bœuf à l'homme se rapportent vraisemblablement, suivant nous, à la teigne tonsurante.

*Gale sarcoptique du mouton.*—Le mouton est assez souvent envahi par une variété de Sarcopte à laquelle on donne le nom de *Sarcoptes scabiei* var. *ovis*. Cet Acarien ne se développe guère que sur les régions dépourvues de laine et en particulier sur la tête : cependant, j'ai vu l'affection se généraliser et s'étendre jusque sur le dos. En raison de son siège habituel, on donne en France à cette forme de gale le nom de noir-museau. Quelques anciennes observations, fort peu précises d'ailleurs, tendaient à faire supposer que la gale du mouton pouvait être transmise à l'homme. En 1858, Delafond, ayant confié aux soins d'un élève de l'École d'Alfort un mouton atteint de gale sarcoptique, vit apparaître sur cet élève une éruption psorique très étendue, qu'on dut traiter après une durée de 49 jours, et qui ne disparut complètement que quinze jours plus tard. En 1877, Gerlach a tenté lui-même, à diverses reprises et avec succès, la transmission de cette gale à un élève, et chaque fois il se vit obligé de traiter cette maladie expérimentale pour en arrêter l'extension. Toutefois, la contagion du noir-museau du mouton à l'homme doit être pratiquement un fait des plus rares, car depuis plusieurs années j'entretiens des animaux affectés de cette maladie, que j'ai transmise à la chèvre et au chien, et aucune des personnes chargées de les soigner ne m'a présenté la moindre éruption.

*Gale sarcoptique de la chèvre.*—Le *S. scabiei*, var. *capræ*, est une forme au moins très voisine de celle qui vit sur le mouton. La gale qu'il détermine débute aussi par la tête, mais ne tarde pas à se généraliser. Walraff, qui l'a vue sévir de 1851 à 1854 dans la vallée

de Prättigau, canton des Grisons (Suisse), a constaté qu'elle se transmettait au cheval, au bœuf, au mouton, au porc et surtout à l'homme; elle se propageait ensuite d'homme à homme; la maladie affectait d'ailleurs un caractère particulièrement grave. A Londres, Henderson a vu, en 1851, la gale de la chèvre se transmettre au cheval et de celui-ci à l'homme; ici encore le prurit était extrêmement violent. Müller, Roloff, Krait ont également vu la gale se transmettre de la chèvre à l'homme.

*Gale sarcoptique du dromadaire.*—Cette forme de spore est due au *Sarcoptes scabiei*, var. *cameli;* elle commence d'ordinaire par les endroits où la peau est mince, et ne tarde pas à se généraliser. La transmission à l'homme a été notée dès 1819 par le médecin Louis Franck, et plus tard par Straus-Durckheim, Hamon, Biett, P. Gervais. Le cas le plus connu est celui de Biett. En 1827, six dromadraires envoyés d'Égypte au Muséum d'Histoire naturelle de Paris furent atteints de la gale. Les gardiens chargés de les soigner contractèrent la maladie, ainsi qu'un palefrenier d'Alfort. "L'éruption avait pris une "si grande intensité chez plusieurs d'entre eux, qu'il survint des "symptômes d'inflammation gastro-intestinale, et chez deux de ces "hommes, vigoureusement constitués, une infiltration générale." En Arabie, d'après Palgrave, on observe souvent aussi le passage de la gale du chameau à l'homme.

*Gale sarcoptique du lama.*—Le Sarcopte du lama serait identique à celui du dromadaire, et la gale qu'il développe présente d'ailleurs les mêmes caractères que celle de ce dernier animal. Delafond et Bourguignon ont constaté, en 1858, la transmission de cette affection à deux élèves de l'École d'Alfort chargés de donner leurs soins à un lama galeux. Il survint des troubles tels qu'on dut traiter la maladie au bout d'un mois.

*Gale sarcoptique du porc.*—Elle est due au *Sarcoptes scabiei*, var. *suis;* elle paraît avoir son siège primitif à la tête et dans les parties supérieures du tronc, mais finit souvent par envahir la totalité du corps. Bateman en Angleterre, Bontekoe et Heckmeyer en Hollande, von Gemmern en Allemagne ont relaté des observations cliniques témoignant de la transmission de la gale du porc à l'homme; tantôt la maladie s'éteignait spontanément au bout d'une douzaine de jours, tantôt elle ne cédait qu'à un traitement approprié. Delafond fut contaminé en disséquant la peau d'un porc galeux; l'affection évolua lentement; mais vers le trentième jour, comme elle menaçait de se généraliser, on dut avoir recours à des frictions médicamenteuses. D'autre part, Siedamgrotzky rapporte qu'à l'École vétérinaire de Dresde, deux élèves s'étant appliqué sur le bras, au moyen d'un bandage, un fragment de peau provenant d'une truie galeuse, il en résulta une gale très prurigineuse qui chez l'un cèda d'elle-même au bout de 48 heures, et qu'on fut obligé de traiter chez l'autre.

Gerlach, de son côté, avait déposé sur la peau de l'homme des Sarcoptes provenant d'un *sanglier*. L'éruption qui se produisit fut peu étendue et faiblement prurigineuse; elle disparut sans intervention au bout de huit à dix jours.

*Gale sarcoptique du chien.* — Le *Sarcoptes scabiei*, var. *canis*, détermine une gale assez commune, débutant le plus souvent par la tête s'étendant avec rapidité et tuant quelquefois les animaux dans l'espace de deux ou trois mois. La contagion de cette gale à l'homme a été établie depuis longtemps par les observations de Chabert, Grognier, Sauvages, Viborg, Mouronval, etc., etc. Dans une épizootie de gale qui a sévi en 1890 sur les chiens, en Allemagne, des centianes de personnes ont été contaminées; dans l'espace d'un mois, Fröhner a constaté, à Berlin, vingt et un cas de cette contagion. Delafond a vu la maladie envahir un élève chargé de soigner un chien galeux; il a pu recueillir, dans les sillons formés sur la main, des Sarcoptes qu'il a reconnus comme identiques à ceux du chien ; au bout d'un mois, on fut obligé de traiter cette gale. Cet observateur a, de plus, déposé sur lui-même et sur trois élèves, des Sarcoptes recueillis sur un chien galeux. Sur deux de ceux-ci, l'éruption a été très fugace. Sur l'autre élève et sur Delafond, la gale s'est bien développée, a persisté six semaines et n'a cédé qu'à un traitement antipsorique. Gerlach a tenté avec succès un essai du même genre.

*Gale sarcoptique du lion.*—Alibert rapporte qu'un préparateur d'anatomie chargé de dépouiller une lionne galeuse contracta la gale, ainsi que l'artiste chargé de l'empailler, le capitaine du bâtiment qui l'avait transportée, le domestique de celui-ci et plusieurs autres personnes qui se trouvaient à bord. Rayer cite un fait analogue. Enfin, Delafond et Bourguignon ont vu de même une éruption psorique survenir chez plusieurs personnes qui avaient un contact journalier avec des lions destinés aux représentations d'un cirque de Paris. Ils trouvèrent le même parasite sur les animaux et sur les hommes. De plus, ils déposèrent sur les bras de quatre personnes des femelles fécondées de Sarcoptes pris sur ces lions ainsi que sur une hyène qui avait gagné leur gale; une éruption psorique se manifesta, mais s'éteignit sans cause appréciable du trentième au quarantième jour, et les quatre sujets guérirent sans traitement.

Mégnin attribue la gale du lion à un Sarcopte identique à celui du loup (*Sarcoptes scabiei*, var. *lupi*), qu'il regarde comme donnant lieu à la variété de psore de l'homme connue sous le nom de *gale norvégienne*.

Nous devons ajouter que nous avons vu nous-même, avec Cadiot, une gale croûteuse du chien présentant avec cette gale de l'homme une grande analogie, et dans laquelle abondaient les Sarcoptes.

Enfin, Rayer a signalé le cas d'un chasseur qui fut atteint d'une gale croûteuse après avoir dépouillé un *renard* galeux.

*Gale sarcoptique du wombat.*—Il ne nous reste plus à signaler, à propos du *Sarcoptes scabiei*, que le fait d'un wombat (*Phascolomys ursinus*) du Muséum d'Histoire naturelle de Paris, qui transmit la gale à son gardien et aux aides-naturalistes chargés de dépouiller le cadavre et d'en préparer la peau.

Une autre espèce de Sarcopte qui vit sur nos animaux domestiques est le Sarcopte nain (*Sarcoptes minor*, Fürstenberg). Il présente deux

variétés ; l'une dite *S. minor*, var. *muris*, qui a pour hôtes le surmulot, le rat d'eau et le coati ; l'autre, de plus petite taille, *S. minor*, var. *cati*, du chat et du lapin. Cette dernière seule nous arrêtera.

*Gale sarcoptique du chat.*—Le Sarcopte nain du chat détermine une gale très grave, qui envahit ordinairement la tête, et ne se généralise qu'à la dernière période. La gale du chat peut se communiquer au cheval, au bœuf, au chien, au lapin et à l'homme. Hertwig rapporte qu'une servante, couchant avec un chat galeux et presque entièrement chauve, ressentit de vives démangeaisons accompagnées d'une éruption sur tout le corps. Berthold cite le cas d'une petite fille qui, ayant laissé un chat galeux reposer sur sa poitrine, éprouva des démangeaisons, puis une éruption. Marrel, Hering, Perroncito, Mégnin, Leonhard ont relaté également des cas de contagion de la gale du chat à l'homme. Enfin, Gerlach a expérimenté sur des élèves de l'École vétérinaire de Berlin et sur lui-même, en déposant sur la peau du bras des croûtes provenant de chats galeux ; il se développa une gale locale, qui cessa d'elle-même au bout de dix à vingt jours. Il semblerait donc résulter de ces expériences que le Sarcopte du chat ne peut s'acclimater chez l'homme, et qu'il ne peut produire qu'une gale éphémère.

On ne possède encore aucun fait établissant que la gale sarcoptique du *lapin* est transmissible à l'homme ; néanmoins, en raison de l'identité de l'Acarien qui la détermine avec celui de la gale du chat, on peut affirmer *à priori* que cette transmission est possible, d'autant que nous avons réussi à transmettre la gale du chat au lapin.

Parmi les autres formes de gale sarcoptique, nous devons encore citer la *gale des pattes* des gallinacés, déterminée par le *Sarcoptes mutans*, Robin. Reynal et Lanquetin, qui les premiers ont étudié cette affection, ont avancé qu'elle pourrait se communiquer à l'homme, mais n'ont pas donné une preuve sérieuse de cette assertion. Les faits qu'ils citent se rapportent plutôt au Dermanysse qu'au Sarcopte changeant. Ils ajoutent bien que des exemplaires de ce Sarcopte, placés sous un verre de montre fixé sur l'avant-bras de l'homme, ont provoqué une éruption vésiculeuse rappelant celle de la gale. Mais les conditions de cette expérience laissent trop à désirer pour qu'on puisse admettre sans réserve la transmission à l'homme de la psore des gallinacés.

On connaît encore, chez les animaux, d'autres formes de gales occasionnées par des Sarcoptidés des genres *Psoroptes* et *Chorioptes* mais il est bien établi à l'heure actuelle qu'elles ne sont nullement transmissibles à l'homme. Les expériences de Delafond et Bourguignon, en particulier, ont montré que si les Psoroptes et les Chorioptes, déposés sur la peau de l'homme, attaquent réllement la peau, ils ne provoquent jamais une gale véritable et succombent dans les 48 heures. Cependant, Schérémétevsky dit avoir vu plus de vingt fois, sur la peau d'individus galeux, le *Chorioptes Symbiotes*, var. *bovis*, que Bogdanoff décrit sous le nom de *Dermatophagoides Scheremetevskyi ;* Zürn l'a aussi trouvé sur la tête d'un homme affecté d'alopécie ; mais il s'agit là sans doute de simples coïncidences, dues à la promiscuité des malades avec les animaux, et

nous ne pensons pas qu'on puisse voir dans les Acariens en question la cause de l'affection dont ces sujets étaient atteints.

En résumé, parmi tous les Sarcoptidés psoriques qui vivent sur les animaux, deux espèces seulement peuvent être considérées comme susceptibles de passer sur l'homme en donnant lieu au développement de la gale : en premier lieu le *Sarcoptes scabiei*, puis, à un moindre degré, le *Sarcoptes minor*.

Avant de quitter le groupe des Acariens, il nous reste encore un type à étudier : c'est le *Demodex folliculorum*, dont les diverses variétés vivent sur l'homme, le chien, le chat, la chèvre, le porc, le mouton, le bœuf, le cheval, etc. Chez le chien, elle donne lieu à une forme de gale très grave, connue tous le nom de *gale folliculaire*. Jusqu'à présent, Zürn est le seul observateur qui ait signalé la transmission de cette affection à l'homme : il dit avoir vu un vétérinaire, un cocher et une femme qui soignaient des chiens atteints de gale folliculaire présenter aux mains et aux pieds une éruption pustuleuse accompagnée d'un violent prurit : les pustules renfermaient des Démodex. C'est là un fait absolument extraordinaire, si l'on songe que, dans les Écoles et les infirmeries vétérinaires, on panse chaque jour, et sans prendre les moindres précautions, des animaux atteints de cette maladie, sans qu'aucun autre cas analogue ait été jamais observé. Bien plus, les essais d'inoculation directe de pus démodécique du chien à l'homme, tentés par Martemucci, Rivolta et Cornevn, ont complètement échoué. Dans un autre sens, Gruby croyait avoir réussi dans un cas à transmettre au chien le Démodex de l'homme ; mais les tentatives expérimentales de Martemucci et Friedberger ont encore échoué.

Faute de détails zoologiques suffisants, on est porté à faire de sérieuses réserves sur la réalité de ces transmissions, car il est possible, que dans l'observation de Zürn, comme dans l'expérience de Gruby, les individus aient été préalablement envahis par leur propre variété de Démodex.

En tout cas, la prophylaxie de la gale découle, de la façon la plus nette et la plus simple, de l'ensemble des faits qui viennent d'être exposés. Il s'agit simplement d'éviter le contact de tous les animaux qui sont affectés ou soupçonnés d'être affectés d'une des deux formes de gale dues au *Sarcoptes scabiei* ou au *Sarcoptes minor*, de ne manier qu'avec précaution les objets sur lesquels les Acariens ont pu être déposés, de traiter rapidement et complètement les sujets atteints ; enfin de désinfecter les locaux qu'ils habitent, la litière, les harnais, etc.

### Parasites internes.

A. *Passant des animaux à l'homme par transmission médiate.*

1ère sect.—Même évolution chez l'homme que chez l'animal.

Coccidium oviforme, perfor. bigeminum.
Lamblia intestinalis.
Balantidium coli.
Tænia serrata ?
Echinorhynchus hominis ?
Echinorhynchus moniliformis.
Ascaris mystax.
Oxyuris vermicularis ?

Tænia canina.
Distoma hepaticum—lanceolatum.
D. truncatum (conjunctum) —D. sinense (foie chat).
Mesogonimus Westermanni (poumon tigre, chien).
Eustrongylus Gigas.
Strongylus paradoxus.
Filaria medinensis — (immitis ?)
Hæmopis.
Linguatula.

2e sect.—Une phase de l'évolution chez l'homme.
Echinococcus.

II.—Parasites internes.—Si l'on en excepte les Hirudinées, auxquelles appartient entre autres l'*Hæmopis sanguisuga*, Moquin-Tandon, les endoparasites animaux sont tous des parasites stationnaires.

Mais la plupart d'entre eux offrent des métamorphoses et des migrations complexes qui ne laissent que peu d'intérêt à la question de périodicité ou de permanence de leur parasitisme. Aussi pensons-nous devoir les classer d'après une autre considération.

Les uns, qu'on pourrait comparer aux parasites temporaires externes, sont simplement communs à l'homme et aux animaux, ou, vivant plus spécialement chez les animaux, peuvent se développer accidentellement chez l'homme. L'homme et les animaux prennent donc ces parasites aux mêmes sources, et la transmission de l'animal à l'homme est le plus souvent médiate; elle a lieu très généralement par l'intermédiaire des boissons ou des aliments que les animaux ont souillés par le dépôt d'œufs ou d'embryons.

Les autres sont toujours transmis directement de l'animal à l'homme, pour accomplir chez celui-ci une des phases de leur évolution; et cette transmission a lieu par la consommation de la chair des animaux infestés.

A.—*Parasites passant des animaux à l'homme par transmission médiate.*—Dans ce groupe, on peut établir deux sections : 1° l'une comprenant les parasites qui suivent chez l'homme une évolution identique à celle qu'ils présentent chez les animaux; 2° l'autre renfermant les parasites qui accomplissent une des phases de leur évolution chez l'homme et l'autre chez l'animal.

1ère *section.*—Pour simplifier l'exposé des faits et éviter la multiplicité de subdivisions toujours un peu arbitraires, nous suivrons simplement ici l'ordre habituel des classifications zoologiques, en partant des Protozoaires pour arriver jusqu'au groupe des Arthropodes.

Les Protozoaires dont nous avons à parler tout d'abord appartiennent à la classe des Sporozoaires : ce sont des Coccidies.

La Coccidie oviforme (*Coccidium oviforme*, Leuck.) vit dans le foie de divers mammifères, notamment du lapin, où un médecin anglais, Hake, l'a trouvée le premier en 1839. Elle se développe, sous la forme d'une petite masse protoplasmique granuleuse, dans les cellules épithéliales des conduits biliaires, et, après s'être entourée d'une coque ou kyste, tombe avec la cellule elle-même dans la lumière du canal. L'accumulation des parasites en certains points de l'organe donne lieu

à des traînées ou à des nodosités blanchâtres, très facilement reconnaissables. Les kystes se débarrassent bientôt des débris des cellules qui les contenaient, grossissent sur place, rassemblent en boule leur contenu et enfin sont expulsés dans l'intestin et emportés avec les fèces. Leur évolution ultérieure doit s'accomplir dans l'eau ou dans la terre humide; au bout de quelques jours, leur contenu se divise en deux, puis en quatre sporoblastes qui s'entourent d'une membrane, constituent ainsi des spores et ne tardent pas à se différencier en deux *corpuscules falciformes* accolés, disposés en sens inverse l'un de l'autre, et accompagnés d'un reliquat protoplasmique.

C'est dans cet état que les kystes, répandus dans les eaux ou sur les aliments, doivent parvenir dans l'organisme; ils se désagrègent dans le tube digestif, mettent en liberté les spores, qui se rompent elles-mêmes et donnent issue aux corpuscules falciformes. Il est probable que ceux-ci, passant à l'état amœboïde, pénètrent alors dans le canal cholédoque jusque dans les conduits biliaires, dont ils vont envahir les cellules épithéliales.

Si le lapin est, comme nous l'avons dit, l'hôte habituel de la Coccidie oviforme, on a cependant trouvé celle-ci dans le foie d'autres mammifères et même de l'homme, ainsi qu'il résulte des observations de Gubler, Virchow, Dressler, Leuckart, etc. Il y a lieu de supposer que l'homme tire ses Coccidies du lapin, et qu'il contracte ces parasites par l'usage d'eaux non filtrées ou de salades souillées par les kystes qu'a rejetés cet animal.

Une autre espèce de Coccidie observée chez l'homme est le *Coccidium perforans*, Leuck., qui évolue de la même manière, quoique plus rapidement, et a son siège spécial dans les cellules de l'épithélium intestinal, où Eimer l'a rencontrée à l'Institut pathologique de Berlin. Or, il existe fréquemment, dans l'intestin du lapin, des Coccidies analogues, et il est encore permis de supposer ici que c'est le lapin qui les transmet à l'homme par l'intermédiaire des aliments et des boissons.

Signalons aussi, dans l'intestin du chien, des Coccidies particulières, découvertes par Rivolta, étudiées ensuite par Railliet et Lucet, et auxquelles le naturaliste américain Stiles à donné récemment le nom de *Coccidium bigeminum*. Elles siègent à l'intérieur et vers la pointe des villosités, et sont remarquables par ce fait qu'on les trouve presque toujours accolées deux à deux. Avec Lucet, nous avons trouvé, dans les fèces d'une femme et de son enfant, tous deux atteints depuis longtemps de diarrhée chronique, des Coccidies offrant à peu près les mêmes dimensions que celles-ci, et qu'il y a peut-être lieu d'assimiler à celles du chien. Ce qui nous porte d'ailleurs à émettre cette opinion, c'est que Kjellberg, à Stockholm, a trouvé précisément dans l'intérieur des villosités, chez l'homme, des Coccidies analogues. Il y a évidemment des recherches à poursuivre de ce côté, d'autant que cette coccidiose est très fréquente chez le chien, et que peut-être l'homme se trouve infesté par cet animal.

La classe des Infusoires nous offre à étudier maintenant deux espèces communes à l'homme et aux animaux : le *Lamblia intestinalis* et le *Balantidium coli*.

Le *Lamblia intestinalis*, Lambl, est un très petit Flagellé qui vit dans l'intestin grêle de divers mammifères, où il se fixe, au moyen d'une large ventouse à bords contractiles, sur les cellules épithéliales des villosités. On le rencontre surtout dans le duodénum et le jéjunum. Dans la première portion du gros intestin, il se montre déjà enkysté, sous l'aspect de corpuscules ellipsoïdes, pourvus d'une enveloppe assez épaisse, comme l'a constaté Perroncito. C'est à cet état d'enkystement qu'il se trouve expulsé avec les fèces, et c'est par l'intermédiaire de ces individus enkystés que s'effectue la transmission du parasite, comme l'ont démontré expérimentalement Perroncito sur la souris, Grassi sur le surmulot, et Calandruccio sur lui-même. Lambl paraît l'avoir observé le premier, en 1859, dans les mucosités gélatineuses de l'intestin des enfants ; il le décrivit sous le nom de *Cercomomas intestinalis*. Depuis cette époque, il a été revu assez fréquemment chez l'homme, en Italie, par Grassi et Perroncito, et peut-être à Calcutta, par Cunningham. Il existe parfois en telle quantité dans l'intestin grêle qu'il recouvre une partie considérable de la muqueuse et arrive, par conséquent, à gêner l'absorption. De plus, d'après Grassi, certaines diarrhées accompagnées d'anémie sont manifestement le fait de ce parasite. On l'a rencontré aussi chez les Muridés (souris, rats, campagnols), chez le chat, le chien, le mouton et le lapin. Ce sont probablement les souris et les rats qui, dans la généralité des cas, le communiquent à l'homme, en souillant de leurs excréments le pain ou les autres substances alimentaires.

Il est donc indiqué de soustraire, autant que possible, les substances en question au contact de ces animaux.

Le *Balantidium coli*, Stein, est un gros Infusoire cilié, mesurant 70 à 100 $\mu$. de long sur 50 à 70 $\mu$. de large, qui a été découvert en 1876 par le professeur Malmsten, de Stockholm, dans les selles d'un homme qui, deux ans auparavant, avait souffert d'une violente attaque de choléra, et qui depuis lors se plaignait de troubles digestifs s'accompagnant alternativement de diarrhée et de constipation. Peu de temps après, cet observateur rencontra le même Infusoire dans le cæcum et le côlon d'une femme qui avait succombé à une colite chronique. Après lui, divers médecins l'ont également retrouvé chez l'homme dans des cas de typhus, de diarrhée, de dysenterie, etc., tant en Suède qu'en Russie, en Italie, en Chine, en Cochin chine, etc. En outre, Leuckart a montré, dès 1863, que le même parasite se rencontre constamment, et en grande abondance, dans le gros intestin du porc, en Saxe. Il a été ensuite observé avec une fréquence variable dans les différentes parties de l'Allemagne, en Suède, en Italie, en Russie et en France, chez le même animal ; à Alfort, nous l'avons trouvé sur tous les porcs examinés (1886) ; Neumann l'a vu également à Toulouse.

Cet Infusoire se reproduit par scission transversale, après conjugaison. D'autre part, quand il a été expulsé de l'intestin avec les excréments, il ne tarde pas à perdre ses cils et à s'enkyster. Or, ce sont ces kystes qui servent à la propagation du parasite ; doués en effet d'une grande force de résistance aux influences extérieures, ils sont emportés par le vent ou par la pluie, et c'est sans doute en avalant l'eau

qui les renferme ou les aliments qui en sont souillés, que le porc s'infeste. Introduits dans le tube digestif, ils résistent à l'action du suc gastrique ; l'Infusoire n'est mis en liberté que dans l'intestin grêle, et passe de là dans le gros intestin, où il se nourrit et se multiplie. Nous devons dire cependant que Calandruccio et Grassi n'ont pu développer le *Balantidium* chez l'homme par l'ingestion de kystes provenant du porc, ce qui leur fait émettre le soupçon d'une différence spécifique entre les parasites de ces deux hôtes. Déjà Wising avait noté que l'Infusoire atteint chez l'homme une taille moins considérable que chez le porc. Quant à son influence pathogénique, nous nous bornerons à dire que, chez le porc, il vit dans un intestin tout à fait sain, tandis qu'on ne l'a encore vu chez l'homme que dans des cas de maladie. En présence de ce fait, et malgré les réserves qui précèdent, il y a lieu de prendre de sérieuses précautions à l'endroit des eaux ou des substances alimentaires qui auraient pu être souillées plus ou moins directement par le fumier de porc.

Nous arrivons à l'examen des parasites qu'on range dans le groupe des Helminthes, groupe assez peu homogène si on ne considère que le côté purement zoologique, mais qu'il convient de conserver si l'on se place spécialement au point de vue de l'hygiène.

Et d'abord, nous avons à mentionner quelques Ténias. Le *Tænia serrata*, Goeze, vit dans l'intestin grêle du chien, et sa larve (*Cysticercus pisiformis*, Zeder) se rencontre dans le péritoine des lièvres et des lapins. D'après Vital, ce Ténia aurait été rencontré deux fois chez l'homme, en Algérie. Comme le dit R. Blanchard, il y a lieu de faire, à l'égard de ces deux observations, les plus expresses réserves. Nous croyons donc inutile d'insister.

Le *Tænia canina*, L. ou *T. cucumerina*, Bloch est aussi, sous sa forme adulte, un parasite de l'intestin grêle du chien. On a longtemps ignoré quel était son hôte à l'état larvaire. C'est un élève de Leuckart, Melnikoff, qui, en 1869, fit connaître les migrations de ce ver. Il reconnut que la larve est un cysticercoïde se développant dans la cavité du corps d'un Ricin parasite du chien, le *Trichodectes canis*. C'est donc en faisant la chasse aux Trichodectes qui vivent sur sa peau que le chien ingère les cysticercoïdes hébergés par ceux-ci et contracte le *Tænia canina*. Pourtant, la plupart des helminthologistes avaient mis en doute la constance de cette migration, en faisant remarquer que le *Trichodectes canis* est un parasite relativement rare, tandis que le *Tænia canina* est des plus communs. Or, Grassi a récemment démontré que la larve de ce Ténia se rencontre habituellement dans la Puce du chien (*Pulex serraticeps*). On doit admettre, par conséquent, que le chien s'infeste surtout en mangeant ses Puces.

Mais le chien n'est pas le seul hôte du Ténia dont il s'agit. Sans parler du *Tænia elliptica* du chat, qui lui est probablement identique, il n'est pas rare de rencontrer le même ver chez les enfants : on en connaît actuellement plus de vingt cas, observés dans les diverses contrées de l'Europe. La raison de ce fait est évidemment la promiscuité dans laquelle vivent si volontiers les enfants et les chiens : il suffit en effet qu'une Puce infestée de cysticercoïdes vienne s'engluer dans les aliments d'un enfant, pour que celui-ci soit exposé à contracter le Ténia.

Et la conclusion qui s'impose au point de vue prophylactique, c'est d'éviter la promiscuité dont nous venons de parler ; on verra du reste dans un instant que d'autres faits parlent dans le même sens.

Le Ténia nain (*Tænia nana*, von Siebold) est un petit ver de 10 à 25 millimètres de long qui a été découvert, en 1851, par Bilharz, au Caire, dans l'iléon d'un jeune homme mort de méningite ; il a été retrouvé depuis, non seulement en Égypte, mais aussi en Amérique, en Angleterre et en Italie.—D'après Grassi, ce ver serait identique au Ténia des Muridés (*Tænia murina*, Dujardin), qui habite l'intestin du surmulot, de la souris, du *Mus pumilus* et du lérot, et qui accomplit sa phase larvaire (*Cercocystis Tæniæ murinæ*) dans la muqueuse même de son hôte définitif. Mais les observations et les expériences de Grassi ne sont pas assez précises pour nous faire admettre cette identité, et nous devons attendre de nouvelles recherches pour être fixés sur le point de savoir si le *Tænia nana* peut être réellement communiqué à l'homme par les petits Rongeurs.

Le même auteur a fourni des données plus précises relativement au *Tænia diminuta*, Rud. (*T. leptocephala*, Creplin), parasite aussi de l'intestin grêle de divers Muridés. Ce ver paraît être réellement identique à celui qui a été décrit chez l'homme sous le nom de *Tænia flavo-punctata*, et qui a été observé à diverses reprises en Amérique et en Italie.

Sa larve, qui est encore un *Cercocystis* (*C. Tæniæ diminutæ*), vit chez un Lépidoptère (*Asopia farinalis* à l'état de chenille et de papillon), chez un Perce-Oreille (*Anisolabis annulipes*) et chez divers Coléoptères (*Axis spinosa*, *Scaurus striatus*). L'hôte habituel serait l'*Anisopia*.

En faisant prendre ces larves à des rats blancs, Grassi et Rovelli ont obtenu le développement du Ténia dans l'intestin. La même expérience a été faite sur deux hommes adultes ; l'un d'eux resta indemne, mais on trouva dans les selles du second, au bout de quinze jours, des œufs identiques à ceux du *Tænia flavo-punctata ;* peu de temps après, l'extrait éthéré de fougère mâle provoqua l'expulsion de nombreux Ténias inermes se rapportant parfaitement à cette espèce.

C'est donc en ingérant avec ses aliments des insectes infestés par les rats que l'homme doit contracter ce parasite. Aussi ne l'a-t-on observé jusqu'à présent que chez des enfants, qui ont la fâcheuse habitude de porter à la bouche tous les corps placés à leur portée.

Si des Cestodes nous passons aux Trématodes, nous trouverons encore une série de parasites communs à l'homme et aux animaux. Nous voulons parler des Distomes.

Le Distome hépatique (*Distoma hepaticum*, L.), plus connu des vétérinaires français sous le nom de *Douve du foie*, se rencontre surtout dans les canaux biliaires du mouton ; mais on peut l'observer aussi chez d'autres mammifères, tels que le bœuf, la chèvre, le chameau, le lama (Delafond), le cheval, l'âne, le cochon, le lapin domestique, le lapin de garenne, le lièvre, le cobaye (Sonsino), etc. On l'a même trouvé chez l'homme. Il est répandu dans toute l'Europe, sauf l'Islande, en Afrique, dans les deux Amérique et en Australie. Sa présence détermine une

affection hydrémique connue sous le nom de cachexie aqueuse : nous croyons avoir démontré d'ailleurs que c'est un véritable suceur de sang. Le ver est hermaphrodite, et émet des œufs qui sont entraînés avec la bile dans l'intestin et évacués avec les excréments. Le développement de l'embryon ne s'achève qu'à l'extérieur du corps de l'hôte, sous l'influence de l'humidité et d'une température modérée. L'embryon sort de l'œuf en soulevant un opercule situé à l'un des pôles ; couvert de cils vibratiles, il nage avec une grande rapidité dans l'eau, jusqu'à ce qu'il ait rencontré l'hôte qui lui convient. D'après les recherches de Weinland, Leuckart et Thomas, cet hôte est un petit Gastéropode d'eau douce, la limnée naine (*Limnæa truncatula*, Müll.). Lorsque l'embryon de Douve a rencontré cette limnée, il enfonce dans ses tissus l'appareil perforateur dont il est muni à son extrémité antérieure, et pénètre ainsi dans la chambre respiratoire du mollusque, où il ne tarde pas à perdre son revêtement ciliaire et à se transformer en une *sporocyste* ovoïde. Celle-ci, qui parfois se multiplie par scission, donne naissance à plusieurs *rédies* qui s'échappent du sac maternel et vont se fixer dans des organes variés de la limnée. Ces rédies donnent elles-mêmes naissance soit à des rédies-filles, soit à des cercaires, c'est-à-dire à des organismes qui offrent déjà les caractères des Distomes, mais possèdent une queue et sont dépourvus d'organes génitaux. Ces cercaires abandonnent le corps du mollusque, nagent dans l'eau ambiante et vont en définitive se fixer sur une plante aquatique, où elles s'enferment, après avoir perdu leur queue, dans un petit kyste protecteur d'une blancheur de neige. C'est donc en consommant ces plantes que les animaux doivent s'infester : le kyste, parvenu dans l'estomac, se désagrège et met en liberté le ver, qui probablement pénètre dans le foie par le canal cholédoque.

Les observations de Distome hépatique chez l'homme, recuillies jusqu'à présent, dépassent une vingtaine ; la plupart se rapportent à des Distomes erratiques rencontrés dans les vaisseaux et dans des tumeurs sous-cutanées. Il faut dire aussi que chez les animaux, les Distomes erratiques ne sont pas très rares, notamment chez le bœuf. L'homme s'infeste évidemment en consommant des plantes sur lesquelles des cercaires se trouvent enkystées, notamment du cresson. Il est donc à recommander de ne pas recueillir, pour l'alimentation, les plantes aquatiques qui croissent dans les endroits fréquentés par les moutons ; dans tous les cas, il convient de les laver, de les nettoyer, de les émonder avec soin, et surtout de se débarrasser de la partie inférieure des tiges, qui est le lieu de prédilection des cercaires.

Le Distome lancéolé (*Distoma lanceolatum*, Rud.) est une espèce de plus petites dimensions, qui habite également les canaux biliaires du mouton et de divers autres herbivores : âne, bœuf, chèvre, porc, lapin, etc. On en connaît jusqu'à présent cinq cas chez l'homme. Au point de vue pathogénique, il paraît avoir une influence analogue à celle du Distome hépatique, mais infiniment moins accusée. Son évolution n'est pas encore bien connue : Willemoes-Suhm avait cru obtenir son développement chez une planorbe (*Planorbis marginatus*) sous la forme de *Cercaria cystophora* ; mais nous avions fait depuis longtemps, avec Ercolani, des réserves expresses sur la réalité du fait, qui est aujourd'hui

absolument controuvé. Piana, de son côté, a considéré le *Cercaria longicaudata*, qui se développe dans l'*Helix carthusiana*, comme représentant l'état larvaire du Distome lancéolé ; et pourtant il est difficile d'admettre *a priori*, en raison du revêtement cilié de l'embryon, que l'hôte intermédiaire soit représenté par un mollusque terrestre. De nouvelles recherches sont donc nécessaires pour déterminer exactement cette évolution, et par conséquent pour établir les mesures prophylactiques qu'il conviendrait de prendre à l'égard de cette espèce.

On connaît encore quelques autres espèces de Distomes qui se rencontrent à la fois chez les animaux et chez l'homme ; mais elles n'ont pour nous qu'un intérêt secondaire.

Ainsi, le *Distoma truncatum*, Ercolani (*Distoma Conus*, Creplin) a été observé dans le foie du chien, du chat, du renard et du phoque, et a reçu des différents observateurs des noms très variés. Il faut en rapprocher le *Distoma conjunctum*, Cobbold, recueilli en 1858 dans les canaux biliaires d'un renard américain (*Vulpes fulvus*) et retrouvé en 1872 par Lewis et Cunningham, à Calcutta, dans le foie du chien paria. McConnell y rapporte un ver qu'il a trouvé à Calcutta, dans le foie de l'homme. Le parasite de Cobbold semble bien identique au *Distoma Conus ;* partant, il est probable que celui de McConnell doit se rattacher à la même espèce.—On n'en connaît pas l'évolution.

Le Distome de Chine (*Distoma sinense*, Cobbold), encore appelé *D. spathulatum*, Leuck., *D. hepatis endemicum* seu *perniciosum*, Baelz, *D. hepatis innocuum*, Baelz, *D. japonicum*, R. Bl., a d'abord été recueilli dans l'Inde et à l'île Maurice, par MacConnell et Macgregor, en 1874–78, dans les canaux biliaires de Chinois qui avaient succombé à des troubles hépatiques paraissant tenir à la présence du parasite. Depuis lors, il a été revu fréquemment au Japon, par divers médecins. D'autre part, en 1816, Ijima a signalé la présence de ce même ver dans le foie du chat ; et nous avons pu voir nous-même à l'Exposition Universelle de Paris, en 1889, des échantillons de Distomes du foie du chat, envoyés par l'École agricole et forestière de Komaba (Japon), sous le nom de *Distoma endemicum*.—Évolution inconnue.

Le Mésogonime de Westermann (*Mesogonimus Westermanni*, Kerbert) était naguère encore classé parmi les distomes, sous les noms de *Distoma Ringeri*, Cobbold, *D. pulmonale*, Baelz. Assez répandu dans l'Asie orientale (Japon, Chine, Corée), il vit en parasite dans le poumon de l'homme, où il a été rencontré pour la première fois, à Formose, par le Dr. Ringer. Mais il avait été déjà observé en 1878, par Kerbert, dans le poumon d'un tigre royal. De plus, il doit se développer aussi chez le chien, car, parmi les échantillons de parasites envoyés par le Japon à l'Exposition de 1889, se trouvaient des *Distoma pulmonale* des bronches du chien. Patrick Manson a suivi l'évolution de cet embryon, et l'a vu sortir de l'œuf pour nager en liberté ; mais on ignore jusqu'à présent quel est le sort ultérieur de cet embryon.(1)

(1) Nous pourrions encore citer ici le Gynécophore hématobie (*Gynecophorus hæmatobius*, Bilharz), qui vit dans la veine porte et les veines du petit bassin, chez l'homme, et que Cobbold a trouvé en outre chez le *Cercopithecus fuliginosus*. C'est le Ver qu'on désigne habituellement sous le nom de Bilharzie.

En somme, on ne connaît encore, en ce qui a trait aux formes précédentes, que l'évolution du *Distoma hepaticum*. Néanmoins, on peut admettre que les autres espèces ont un mode de développement analogue, et recommander dans tous les cas d'éviter l'usage d'eaux ou de plantes aquatiques provenant des localités que fréquentent les animaux susceptibles de posséder ces parasites.

En quittant le groupe des Trématodes, nous nous trouvons en présence de l'ordre des Acanthocéphales, dont nous n'aurons que quelques mots à dire.

En 1857, Lambl, médecin de Prague, faisant l'autopsie d'un enfant de 9 ans, mort de leucémie, trouva dans l'intestin grêle un Acanthocéphale qu'il décrivit sous le nom d' *Echinorhynchus hominis*. Les auteurs qui ont examiné la description et la figure de Lambl ont cherché à rattacher ce ver à des espèces déjà connues. Ainsi, Ant. Schneider était d'avis qu'il s'agissait de l' *Echinorynchus Gigas*, Goeze, parasite habituel du porc, dont la larve vit, d'après lui, dans la larve du hanneton, et d'après Kaiser, dans celle de la cétoine. Leuckart, au contraire, tend plutôt à l'assimiler à *Echinorynchus augustatus*, Rud., espèce assez commune chez les poissons d'eau douce, ou à *Echinorhynchus spirula* Olfers, qui se voit chez certains singes. En présence de ces divergences, il n'y a pas lieu de nous arrêter plus longtemps sur ce parasite.

Grassi et Calandruccio ont étudié récemment une autre espèce d'Échinorynque qui habite l'intestin grêle du surmulot et du loir : il s'agit probablement, à leur avis, de l'*Echinorhynchus moniliformis*, Bremser, que Diesing avait déjà rencontré en Autriche chez le campagnol des champs et le hamster. C'est un coléoptère assez répandu, le *Blaps mucronata*, Latr., qui héberge sa larve, et dans quelques cas ces auteurs ont trouvé plus de 100 larves dans le même blaps. Or, le 26 Décembre 1887, un certain nombre de ces larves furent administrées à un rat blanc ; Calandruccio ingéra le reste. Le 10 Janvier suivant, on retrouvait dans l'intestin du rat un grand nombre d'Échinorynques, mesurant 1 centimètre de longueur. Le 15 Janvier, Calandruccio fut pris de violentes coliques, accompagnées d'un peu de diarrhée, de bourdonnement dans les oreilles, de fatigue et de somnolence. Le 1er Février, il trouva pour la première fois dans ses fèces quelques œufs d'Échinorynques. Le 13 Février, les douleurs abdominales devinrent tellement intenses qu'il se vit forcé de prendre un anthelminthique : il rendit alors 53 Échinorynques bien développés. L'année précédente, les deux auteurs italiens avaient trouvé dans les fèces d'une jeune Sicilienne des œufs d'Échinorynque qu'ils pensent pouvoir rapporter à cette espèce ; mais cette observation est demeurée incomplète. En tout cas, l'expérience précédente est des plus instructives, puisqu'elle démontre que l'*Echinorhynchus moniliformis* des petits rongeurs peut évoluer dans l'organisme de l'homme. A la vérité, les conditions de ce développement doivent être bien rarement remplies, car il faut supposer à un homme une forte dose de distraction pour admettre qu'il puisse ingérer un blaps, c'est-à-dire un insecte de la taille d'un hanneton. Disons cependant qu'on a

signalé une douzaine de cas de ce genre, mais dans lesquels les insectes étaient rendus sans avoir été digérés. D'autre part, les femmes égyptiennes, au dire de Fabricius, mangent des *Blaps sulcata* cuits dans le beurre, en vue de se donner de l'embonpoint.

L'ordre des Nématodes nous offre à considérer des formes un peu plus nombreuses et plus variées.

En tête de celles-ci se placent les Ascarides. L'*Ascaris mystax*, Rud. est commun dans l'intestin grêle du chat, du chien, et de divers carnivores sauvages. De plus, quelques observations recueillies par Pickells, Bellingham, Leuckart, Cobbold, etc. démontrent qu'il peut se rencontrer aussi chez l'homme. Il subit un développement direct, sans hôte intermédiaire. L'embryon se forme lorsque l'œuf est maintenu dans un millieu humide ; la sécheresse suspend son évolution, bien qu'elle ne détruise sa vitalité qu'au bout d'un temps assez long ; une fois formé il demeure ordinairement dans la coque. Les essais d'infestation directe n'ont pas jusqu'à présent donné de résultats réellement positifs ; néanmoins toutes les observations qu'on a pu faire plaident en faveur d'un développement direct. Dans ces derniers temps, Grassi a ingéré à diverses reprises des Ascarides du chat sans parvenir à les garder vivants dans son tube digestif ; il a pris de même, avec Calandruccio, des œufs embryonnés de l'Ascaride du chien, avec un résultat complètement négatif. Aussi tend-il à admettre que l'occurrence de ce ver chez l'homme est au moins douteuse. Il est bien difficile d'accepter une telle conclusion, contraire aux faits cités plus haut, d'autant plus que le mode d'infestation des animaux eux-mêmes offre encore certaines obscurités. En tout cas, il nous paraît y avoir ici un motif de plus pour l'homme d'éviter la promiscuité avec le chien.

Divers auteurs ont admis que l'*Ascaris lumbricoides*, L., de l'homme, était quelquefois aussi parasite du bœuf ; mais les recherches de Neumann ont montré que l'Ascaride du bœuf constitue réellement une espèce à part (*Ascaris vitulorum*, Gœze). De même, Dujardin a montré que l'Ascaride du porc, qu'on a également voulu identifier à l'Ascaride lombricoïde, représente une espèce non moins distincte (*Ascaris suilla*, Duj.).

Nous ne ferons qu'une simple mention de l'*Oxyuris vermicularis*, L., parasite du gros intestin de l'homme. D'après Zürn, ce ver se rencontrerait exceptionnellement chez le chien. Peut-être s'agit-il plutôt de l'*Oxyuris compar*, trouvé par Leidy dans l'intestin grêle du chat.

L'Eustrongle géant (*Eustrongylus Gigas*, Rud.), le plus grand des Nématodes, se développe ordinairement dans les reins. Il est surtout fréquent chez les mammifères ichtyophages, tels que la loutre, le vison d'Amérique, la phoque, etc. ; mais on le rencontre aussi chez d'autres carnivores, comme le chien, le loup, le putois, la martre, et même chez des herbivores, tels que le cheval et le bœuf. Enfin, on possède plusieurs observations authentiques relatives à sa présence chez l'homme. Les phases de son évolution sont encore inconnues ; toutefois Schneider et Leuckart admettent, avec beaucoup de vraisemblance, que cette évolution doit comporter le passage par un hôte intermédiaire représenté par quelque espèce de poisson.

Diesing a donné le nom de *Strongylus longevaginatus* à un ver trouvé en 1845 par le Dr. Jortsits, de Klausenbourg en Transylvanie, dans le poumon d'un enfant de six ans, mort de maladie inconnue. Leuckart, qui en reçut de Diesing deux exemplaires, est d'avis que ce ver n'est autre que le *Strongylus paradoxus*, Mehlis, hôte habituel des voies respiratoires du porc. En 1888, J. Chatin a communiqué à l'Académie de Médecine de Paris des exemplaires de cette dernière espèce, trouvés dans les déjections d'un malade atteint de troubles gastro-intestinaux, malade qui faisait, durant une partie de l'année, un grand commerce de viande fraîche de porc. Mais il est probable qu'il s'agit ici d'un cas de pseudo-parasitisme, la présence du ver dans le tube digestif étant un fait anormal, qui ne peut guère s'expliquer que par l'ingestion directe de vers contenus dans la viande. On ne connaît pas encore l'évolution de ce Strongle paradoxal; mais tous les essais d'infestation directe tentés jusqu'à présent sur le porc sont demeurés sans résultat. Il est possible que sa larve doive passer par un hôte intermédiaire.

Un autre ver fort intéressant est la Filaire de Médine (*Filaria medinensis*, Velsch), dont on ne connaît jusqu'à présent que la femelle Cette Filaire est propre aux pays chauds : elle est commune en Afrique de puis la côte de Guinée (ce qui lui a valu le nom anglais de *Guinea-worm*) jusqu'en Égypte; en Asie, elle est répandue en particulier dans presque toute l'Arabie; enfin, elle est devenue endémique dans diverses localités de l'Amérique du Sud. On la connaît surtout comme parasite de l'homme, dont elle envahit d'ordinaire le tissu conjonctif sous-cutané; mais elle se rencontre aussi chez divers animaux. D'après Avenzoar et de Marchais, elle s'attaquerait fréquemment au bœuf; Clarkson, Fleming, Burke l'ont observée chez le cheval, dans l'Inde; d'autres observateurs l'ont vue chez le chien, en Amérique, dans l'Inde et en Égypte, puis chez le guépard, le *Canis lupaster* et le chacal. Chez les carnivores, elle paraît même se montrer en plus grand nombre que chez l'homme. Et, fait curieux à noter, elle attaque fréquemment ces animaux dans la Basse-Égypte, tandis qu'elle ne semble pas, jusqu'à présent, s'être acclimatée chez les indigènes de cette région. Les migrations de cette espèce ont été déterminées par Fedtchenko. Les embryons émis par la Filaire doivent parvenir dans l'eau : ils pénètrent alors par effraction dans la cavité du corps de petits crustacés d'eau douce appartenant au genre *Cyclops*, et y subissent une mue qui les amène à l'état larvaire. On est porté à admettre qu'ils doivent alors réintégrer l'organisme de l'homme ou des animaux lorsque ceux-ci boivent l'eau contenant les cyclopes, ces crustacés passant inaperçus en raison de leur taille exiguë. Cependant, Fedtchenko a tenté sans succès d'infester deux jeunes chiens et un chat, en leur faisant prendre dans du lait et de l'eau des cyclopes remplis de larves. Il est indiqué, en tout cas, dans les régions où se rencontre la Filaire de Médine, de ne faire usage que d'eau filtrée.

Mentionnons en outre, dans le même genre, la Filaire cruelle (*Filaria immitis*, Leidy), qui vit dans le cœur droit et les artères pulmonaires du chien, plus rarement dans le tissu conjonctif sous-cutané ou intermusculaire. Rivolta paraît l'avoir vue aussi chez le

renard, et Bowlby l'a signalée récemment chez l'homme, mais ces observations auraient besoin d'être contrôlées.—Évolution inconnue.

Les *Hæmopis*, qui se présentent maintenant à notre examen, n'appartiennent plus au groupe des Helminthes : ce sont des Annélides.

L'Hémopis sanguisugue (*Hæmopis sanguisuga*, Moquin-Tandon) (1), connue en France sous les noms vulgaires de *Sangsue de cheval* et de *Voran*, se rencontre çà et là dans le centre et le nord de l'Europe ; mais elle est plus commune dans le midi, et se montre infiniment plus abondante encore dans le nord de l'Afrique, depuis le détroit de Gibraltar jusque sur les côtes de Syrie. Cette Sangsue vit dans les mares, les fossés et les petites sources, où elle se reproduit. Son développement est direct. Contrairement à ce que nous avons vu pour les parasites internes qui précèdent, elle ne présente en réalité qu'un parasitisme temporaire : sa nourriture se compose en effet de sang, mais comme ses mâchoires sont trop faibles pour percer la peau des mammifères, elle cherche à pénétrer dans les cavités naturelles pour s'attaquer aux muqueuses. C'est ainsi qu'on la rencontre fréquemment dans la bouche, l'arrière-bouche, le larynx, les fosses nasales, etc., des chevaux, des mulets, des bœufs, des chameaux, et des autres animaux qui vont à l'abreuvoir. L'homme lui-même est sujet à ses attaques lorsqu'il boit sans précautions dans les sources. Une fois gorgée, la Sangsue peut se détacher et se laisser glisser dans l'eau quand l'animal retourne à l'abreuvoir.

Le dernier parasite que nous ayons à examiner dans cette section est un Arthropode, la Linguatule rhinaire (*Linguatula rhinaria*, Pilger, *Pentastoma tænioides*, Rud.). À l'état adulte, cette espèce vit dans les cavités nasales du chien, du loup, du renard, plus rarement dans celles du cheval, du mulet et de la chèvre. Une seule fois on l'a rencontrée chez l'homme. Son évolution est assez compliquée. Les œufs, déposés en nombre immense, par les femelles, dans les fosses nasales, sont expulsés avec le mucus, surtout par le fait des éternuements de l'hôte. Ils peuvent se trouver rejetés dans les flaques d'eau, sur l'herbe des prairies et en général sur les aliments des herbivores : repris par ces animaux, ils arrivent dans l'estomac, où la coque se trouve détruite sous l'influence du suc gastrique. L'embryon, qui s'était formé avant la ponte, est ainsi mis en liberté ; il traverse la paroi de l'intestin et va se fixer dans les ganglions mésentériques, dans le foie ou dans le poumon. Là, il subit neuf mues successives, qui exigent environ 23 semaines ; après quoi il est à l'état de larve définitive, forme qu'on désigne encore aujourd'hui sous le nom de Linguatule denticulée (*Linguatula serrata*, Frölich). Cette larve a été fréquemment observée dans les viscères, et en particulier dans les ganglions mésentériques, le foie et le poumon d'un grand nombre de mammifères : mouton, bœuf, chèvre, chameau, cheval, chat, lapin, lièvre, cobaye, surmulot, etc., etc.

---

(1) Des recherches récentes de R. Blanchard, il résulte que l'Hirudinée signalée sous ce nom comme s'attaquant à l'homme et aux animaux en Afrique, n'est autre que *Limnatis nilotica*, Sav., et que *Hæmopis sanguisuga*, Bergmann, d'Europe, est identique à *Aulastoma gulo*, Moquin-Tandon.

Elle a même été trouvée assez souvent chez l'homme, d'abord par Zenker à Dresde, puis par d'autres observateurs, dans diverses parties de l'Allemagne, en Suisse, en Russie, en Autriche. Il n'est pas impossible que ces larves arrivent quelquefois, en traversant les bronches, à gagner les cavités nasales de leur hôte ; Gurlt a trouvé, en effet, des larves libres dans la trachée d'un lièvre et d'une chèvre. En général, cependant, la migration définitive s'effectue d'une façon quasi-passive, et les larve sont condamnées à périr si les viscères de leur hôte ne sont pas dévorés, en temps voulu, par un carnassier. Mais que ces viscères soient abandonnés à un chien, par exemple : les larves, mises en liberté par la déchirure des tissus, tendront à gagner immédiatement les cavités nasales, par la voie des narines ou des orifices gutturaux. Elles y subiront une nouvelle et dernière mue, et parviendront ainsi à l'état adulte. On a vu que l'homme peut héberger et la larve et la forme adulte. Il contracte évidemment la première par l'ingestion d'aliments ou de boissons souillés par les œufs que laissent d'ordinaire échapper les chiens ; quant au mode suivant lequel la forme adulte se développe dans les cavités nasales, on peut supposer qu'il s'agit, soit d'une ingestion de viande crue, soit d'une migration directe dans les voies respiratoires de larves fixées dans le poumon.

2° *section.*— On se rappelle que cette section doit comprendre les parasites qui, transmis des animaux à l'homme par voie médiate, doivent accomplir une des phases de leur évolution chez ceux-là et l'autre chez celui-ci.

Tel est le cas du *Tænia Echinococcus*, von Siebold. C'est un petit ver de trois à quatre millimètres de long, formé seulement de trois ou quatre anneaux, qui habite l'intestin grêle du chien et de quelques autres carnivores. Son état larvaire ou hydatique est représenté par l'*Echinococcus polymorphus*, Diesing, dont l'habitat est des plus variés, car on en a signalé la présence chez l'homme, divers singes, le lapin, le porc, le cheval, le zèbre, le bœuf, le mouton, la chèvre, le chameau, etc. Il est certain que les animaux herbivores trouvent surtout dans les pâturages les œufs d'où dérivent les Échinocoques ; pour ce qui a trait à l'homme, il faut plutôt incriminer les eaux non filtrées, sans oublier cependant que les salades, les fruits tombés à terre, les aliments et les ustensiles de cuisine souillés au contact direct du chien sont aussi des sources directes d'infestation dont il faut savoir tenir compte à l'occasion.

B. *Parasites internes passant des animaux à l'homme par transmission immédiate.*

| | |
|---|---|
| *Tænia Solium.* | *Bothriocephalus latus.* |
| *Tænia saginata.* | *Trichina spiralis.* |

*B.—Parasites passant des animaux à l'homme par transmission immédiate.*—Il s'agit ici de parasites bien connus, dont l'évolution, déterminée par des expériences très nombreuses et très précises, est devenue pour ainsi dire de connaissance vulgaire, et sur lesquels, par conséquent, nous n'aurons pas à nous étendre à ce point de vue. Quant

aux conditions de la prophylaxie, elles sont également bien établies à l'heure actuelle, et il nous suffira d'en faire un exposé succinct.

Le premier de ces parasites qui s'offre à nous est le *Tænia Solium*, qui habite, à l'état rubanaire, l'intestin grêle de l'homme, et dont les anneaux sont rejetés souvent en petits chaînons dans l'acte de la défécation. Il est facile de le reconnaître à sa tête armée d'une double couronne de crochets, à ses orifices génitaux assez régulièrement alternes, aux branches de la matrice épaisses et peu nombreuses. A l'état vésiculaire, ce Ténia est réprésenté par le *Cysticercus cellulosæ*, Rud., qui se montre sons l'aspect d'une vésicule généralement ellipsoïde, de 6 à 20 mm. de long sur 5 à 10 de large, offrant vers le milieu de sa longueur une tache blanche qui correspond à la tête invaginée. L'hôte habituel de ce cysticerque est le porc domestique; mais on l'a rencontré aussi chez le sanglier, le chien, le chat, le chevreuil, le rat noir, etc., et l'on peut ajouter qu'il n'est pas rare chez l'homme.

Il siège dans le tissu conjonctif de la plupart des organes, principalement des muscles, et sa présence dans l'organisme caractérise la maladie depuis longtemps connue sous le nom de *ladrerie*. Nous n'avons pas à présenter ici l'histoire complète de cette affection, dont la connaissance remonte à une haute antiquité. Nous rappellerons seulement qu'elle est assez difficile à distinguer du vivant de l'animal, et qu'on arrive seulement à la certitude de son existence par le *langueyage*, c'est-à-dire par l'examen direct de la langue. Même sur l'animal mort, il est parfois difficile de reconnaître la présence des cysticerques, lorsque la ladrerie est peu prononcée; mais on sait qu'ils ont pour siège privilégié les muscles de la face profonde de l'épaule, ceux du cou, la portion charnue du diaphragme, etc. Il faut en outre savoir que le charcutier peut dissimuler l'état de la viande envahie, par l'énucléation des cysticerques de la surface. Mais où la difficulté devient surtout considérable, c'est dans l'examen des préparations de charcuterie faites avec la viande hachée : il y a lieu alors de recourir au procédé recommandé par Schmidt-Mulheim, qui permet de les mettre en évidence par suite de leur résistance à l'action du suc gastrique.

Puisqu'il est bien établi que l'usage de la viande de porc ladre donne lieu chez l'homme au développement du *Tænia Solium*, quelles sont les mesures d'ordre prophylactique qu'il convient de prendre à l'égard de cette viande ? Nous ne pouvons et ne devons les résumer en quelques mots.

1°. Chercher tout d'abord à diminuer la fréquence de la ladrerie par la séquestration des animaux dans les porcheries, et l'alimentation avec des substances préalablement soumises à l'action d'une température élevée. Si les porcs doivent être nécessairement conduits au pâturage ou à la glandée, répandre parmi les populations de la campagne des notions sur le danger de la dissémination des excréments humains.

2°. Soustraire autant que possible à la consommation, au moyen d'une inspection rigoureuse, la chair des porcs atteint de ladrerie à un degré quelconque.

Comme un certain nombre de sujets échapperont toujours à cette inspection, recommander de ne manger la viande de porc qu'après l'avoir

soumise à une cuisson prolongée et complète. Les recherches de Perroncito ont établi que le cysticerque meurt d'une façon certaine s'il est maintenu plus d'une minute à la température de 50°, et celles de Küchenmeister et autres montrent que des morceaux de viande assez épais, soumis à l'ébullition pendant quelques heures, atteignent cette température jusque dans leur partie centrale. Quant à l'influence de la salaison et du fumage, elle doit être assez prolongée pour détruire définitivement la vitalité des cysticerques.

En France, les mesures de police sanitaire applicables à la ladrerie sont laissées, d'après la loi municipale du 5 Avril 1884, à l'appréciation de l'autorité communale, qui admet une tolérance variable, mais généralement fâcheuse, en ce sens que la consommation des viandes modérément envahies étant autorisée, les cas de *Tænia Solium* tendent à se multiplier, les viandes en question éveillant peu de défiance chez le consommateur.

Il est à remarquer toutefois que dans beaucoup de localités, et en particulier à Paris, la fréquence du *Tænia Solium* accuse une diminution très accusée, en raison de l'habitude qui s'est répandue dans le public de ne manger la viande de porc qu'après une cuisson convenable.

Le *Tænia saginata*, également parasite de l'homme, se distingue du précédent, d'abord parce que ses anneaux, un peu plus grands et plus vivaces, sont émis isolément et dans l'intervalle des selles, puis par sa tête inerme, ses orifices génitaux très irrégulièrement alternes, et enfin les branches de la matrice minces et nombreuses. Son cysticerque (*Cysticercus bovis*, Cobbold) se développe surtout chez les bêtes bovines; mais on l'a rencontré aussi chez la girafe et peut être même chez l'homme. Zenker en a communiqué expérimentalement à la chèvre, Heller à la chèvre et au mouton. L'histoire de la ladrerie du bœuf, plus récemment élucidée que celle de la ladrerie porcine, présente en somme les mêmes traits généraux. La maladie offre cependant, en général, ce caractère d'être plus discrète que chez le porc, ce qui, à notre avis, est la cause principale de la prédominence qui tend à se manifester, dans beaucoup de localités, du *Tænia saginata* sur le *Tænia Solium*, les animaux affectés échappant plus facilement à l'inspection, aussi bien après qu'avant la mort. Sous ces réserves, se que nous avons dit de la ladrerie du porc peut s'appliquer à la ladrerie du bœuf, et il nous paraît inutile de nous étendre d'avantage sur cette affection. Nous relèverons seulement ce fait que les muscles masséters, et en particulier les internes ou ptérygoïdiens, sont le principal lieu d'élection des cysticerques, de sorte que l'examen spécial de ces muscles s'impose aux inspecteurs des viandes : depuis que cette règle est adoptée à l'abattoir central de Berlin, la découverte des bœufs ladres est infiniment plus fréquente qu'autrefois. Cest une mesure qui devrait devenir obligatoire dans les différents états de l'Europe.

A côté des Ténias se placent les Bothriocéphales, dont une espèce le *Bothriocephalus latus*, Bremser, est assez commune chez l'homme, du moins dans certaines régions, telles que la Suisse française, la Bavière, la Haute-Italie, les provinces russes de la Baltique, et, en dehors de l'Europe, dans le Turkestan et au Japon. Ce n'est point à dire qu'il ne vive que dans ces contrées, mais il est certainement beaucoup plus

rare ailleurs. On l'a signalé du reste chez le chien et chez le chat, où il acquiert des dimensions moindres que dans l'intestin de l'homme. Jusqu'à ces derniers temps, l'origine de ce ver était demeurée assez obscure. Sa fréquence dans le voisinage des lacs avait fait penser, il est vrai, qu'il devait vivre, pendant son jeune âge, chez un animal aquatique. Mais c'est seulement en 1881–82 que la question fut résolue expérimentalement. Max Braun démontra que la larve ou plérocercoïde, d'aspect vermiforme et non vésiculeuse, se développe chez le brochet (*Esox lucius*), où on la trouve enkystée non seulement dans les viscères, mais aussi dans les muscles. Depuis lors, des recherches poursuivies dans le même sens ont démontré que d'autres espèces de poissons jouent également le rôle d'hôte intermédiaire, par exemple la lotte (*Lota vulgaris*), la perche (*Perca fluviatilis*), et plusieurs salmonidés (*Salmo Umbla, Trutta vulgaris, Trutta lacustris, Thymallus vulgaris*) ; au Japon, ce rôle est rempli par *l'Onchorhynchus Perryi*.

C'est donc en mangeant la chair insuffisamment cuite des poissons en question que l'homme contracte le Bothriocéphale : aussi ce ver est-il relativement répandu chez les pêcheurs et en général dans les populations ichtyophages. Partant, les indications prophylactiques sont des plus simples et se résument en ces deux points : empêcher le déversement des excréments humains dans les lacs ou dans les rivières ; ne consommer la chair des poissons qu'après une cuisson complète(1).

Il ne nous reste plus à parler que de la Trichine (*Trichina spiralis*, Owen) et de la trichinose. Il est presque superflu de rappeler que les Trichines arrivent ordinairement à l'état adulte, sexué (Trichines intestinales) dans l'intestin grêle des mammifères, et que les larves (Trichines musculaires) auxquelles elles donnent naissance émigrent dans les muscles de leur hôte, où elles s'enroulent en spirale et s'enkystent. Une fois enkystées de la sorte, ces larves ne peuvent parvenir à l'état adulte qu'autant qu'elles sont ingérées, après avoir acquis un certain développement, par un autre animal à sang chaud. On est parvenu à infester expérimentalement les muscles d'un grand nombre de mammifères ; mais l'infestation spontanée n'a été observée jusqu'à présent que chez l'homme, le porc, le sanglier, l'hippopotame, le hamster, le rat noir, le surmulot, la souris, le chien, le chat, le renard, le raton, la martre et le putois. L'homme tire presque toujours ses Trichines du porc, et celui-ci doit les prendre principalement aux petits rongeurs. Les rats et surtout les surmulots paraissent être les hôtes primitifs de ces parasites, qu'ils contractent en s'entre-dévorant.

D'après ces données, résumées d'une facon aussi sommaire que possible, la prophylaxie de la trichinose doit évidemment comporter, comme celle du téniasis et de la bothriocéphalose, deux indications essentielles, très simples en apparence, mais en réalité soulevant de fort

(1) Je laisse de côté le *Bothriocephalus cordatus*, Leuckart, trouvé au Groenland chez l'homme, le chien, le phoque barbu et le morse, ainsi que le *Bothriocephalus Mansoni*, Cobbold, assez commun au Japon et en Chine, chez l'homme, et probablement identique au *Sparganum reptans*, Diesing, observé chez divers animaux : l'évolution de ces formes est en effet, jusqu'à présent, totalement inconnue.

graves difficultés pratiques : 1° enrayer la propagation de la trichinose chez le porc ; 2° empêcher l'infestation de l'homme par le porc trichiné.

Nous avons dit que les rats doivent être les premiers propagateurs de la trichinose. Il n'est pas rare, en effet, de rencontrer des Trichines dans les muscles de ces animaux. En Allemagne et en Autriche, Leisering en a trouvé chez plus de 8 pour 100. En France même, où la trichinose est excessivement rare, Vulpian et Laboulbène en ont observé chez des rats d'égout, et Colin en a fréquemment trouvé à Alfort. Or, on sait que le porc mange volontiers des rats à l'occasion. Kuhn a d'ailleurs constaté la réalité de ce fait. On pourrait donc espérer qu'en exterminant les rats, on détruirait, par le fait même, la Trichine. Il est vrai que l'extermination des rats est chose au moins très difficile à réaliser ; et d'autre part Zenker et Gerlach ont soutenu, avec quelque apparence de raison, que les rats pourraient bien n'être si fréquemment infestés que précisément parce qu'ils mangent souvent de la viande de porc trichiné. Au surplus, il est certain que le porc s'infeste souvent en mangeant lui-même de la viande de porc : c'est le cas qui se présente d'ordinaire dans les clos d'équarrissage et dans les abattoirs. L'enseignement qui ressort de tous ces faits, c'est la nécessité de surveiller rigoureusement l'alimentation des porcs, de les nourrir de préférence de substances végétales, d'éviter qu'ils puissent se repaître d'excréments humains, de résidus de boucherie, et surtout de cadavres de rats ; de ne leur distribuer enfin de substances animales qu'après avoir soumis celles-ci à une cuisson suffisante.

Quant à la préservation directe de l'homme, elle doit être basée tout d'abord sur la détermination du degré de vitalité des Trichines. On sait depuis longtemps que ces parasites survivent à leur hôte et résistent fort bien à la *putréfaction*, puisque au bout de trois mois on peut encore les trouver vivantes dans la viande. La *salaison* les tue en général plus ou moins rapidement, mais il n'y a rien d'absolu à cet égard, et on en trouve encore de parfaitement vivantes dans des jambons américains importés depuis plus d'un an. Le *fumage*, quoique paraissant un peu plus efficace, est loin de suffire dans tous les cas à rendre les viandes inoffensives. Reste l'action des hautes et des basses températures. On n'est pas absolument d'accord sur l'influence du *froid*. Bouley et Gibier ont vu les Trichines périr en soumettant deux gros morceaux de jambon, pendant deux heures et demie, à une température de 22 à 27 degrés ; mais Leuckart a constaté la présence de Trichines vivantes dans des jambons frais exposés pendant trois jours à une température de 22 à 25 degré Du reste cette question n'a pas une grande importance pratique, car ces expériences se placent en dehors des conditions usuelles de l'économie domestique. Il n'en est plus de même en ce qui se rapporte à l'influence de la *chaleur*. La plupart des expérimentateurs estiment qu'une température de 70° suffit à faire périr les larves enkystées ; les exceptions à cette règle peuvent être négligées. Mais les parties centrales des morceaux de viande n'atteignent cette température qu'après un temps plus ou moins long, variant avec leur volume et leur mode de cuisson. Pour les viandes bouillies, par exemple, il faut prolonger l'*ébullition* pendant une demi-heure au moins par chaque kilo-

gramme. Les viandes rôties sont toujours plus dangereuses, car la couche extérieure *saisie* par la cuisson retarde la pénétration de la chaleur dans les parties profondes. Dans la pratique, on juge que la cuisson est suffisante lorsque la viande a perdu sa couleur rouge, et qu'il ne s'écoule plus de jus saignant sur une coupe. C'est donc en somme la cuisson qui semble devoir constituer la plus sérieuse garantie relativement à la préservation directe de la trichinose; encore faut-il qu'elle soit réellement complète. Aussi est-ce surtout aux habitudes culinaires de ses habitants que la France doit l'heureuse circonstance de n'avoir eu à enregistrer, jusqu'à présent, qu'une seule petite épidémie de trichinose.

Mais il existe des contrées, comme l'Allemagne et les États-Unis, où la trichinose règne en permanence, et où il y a lieu, par conséquent, de prendre des mesures spéciales de police sanitaire pour éviter la transmission du parasite à l'homme. Ces mesures sont sans doute du même ordre que celles dont nous avons parlé à l'occasion de la ladrerie, mais elles doivent être plus rigoureuses encore, étant donnée la haute gravité de la trichinose chez l'homme. Nous ne croyons pas devoir faire ici l'examen détaillé des règles qui doivent être suivies dans l'inspection des viandes trichinées; il nous suffira de rappeler l'organisation adoptée en Allemagne, où, à l'exemple du Duché de Brunswick, un grand nombre de villes ou d'états ont institué un service complet d'inspection de la viande de porc, et où une véritable armée d'experts, hommes et femmes, est employée à ce contrôle. Ces mesures sont complétées, du reste, par toute une série de lois, décrets et arrêtés, qu'il nous est impossible de passer en revue, et qui ont en somme la même base que les règles relatives aux maladies d'origine microbienne. Les lois en question, comportant souvent des peines afflictives ou infamantes pour qui les transgresse, ont pu paraître à certains esprits revêtir un caractère trop rigoureux; mais il ne faut pas oublier pourtant que les gouvernements ont le droit et le devoir de mettre en œuvre tous les moyens propres à sauvegarder la vie et la santé des citoyens. Et c'est dans le même ordre d'idées qu'il faut comprendre les mesures que telle nation, jusqu'alors indemne, est appelée parfois à prendre à l'égard d'un autre pays dont les importations constituent pour elle un danger permanent.

## Conclusions.

Maintenant que nous avons épuisé à peu près complètement cette longue liste des parasites transmissibles des animaux à l'homme, il convient de jeter sur l'ensemble un coup d'œil rétrospectif, et de voir s'il n'est pas possible de dégager des faits acquis certaines indications générales applicables à la prophylaxie.

A la vérité, tous les moyens préventifs que l'on peut opposer au parasitisme ont pour effet de protéger l'homme contre la transmission des parasites provenant des animaux, puisqu'ils concourent à enrayer la multiplication de ces parasites. Or, en prenant ce point de départ même, il faut reconnaître que la lutte contre ces êtres n'est réellement efficace qu'autant qu'on est fixé sur leur évolution propre, ce qui revient à dire que les indications prophylactiques sont beaucoup plutôt d'ordre

spécial que d'ordre général. Mais on a vu que, pour un bon nombre des parasites qui peuvent passer des animaux à l'homme, les premières phases de l'évolution, c'est à dire celles qu'il importe le plus de connaître, sont à peine soupçonnées ou sont même encore totalement inconnues. Néanmoins, nous ne sommes pas entièrement désarmés vis-à-vis de ceux-ci. Sachant, en effet, que les parasites dérivent, comme tous les êtres vivants, d'individus ayant subi la même évolution qu'eux-mêmes sont appelés à subir, on pourra s'opposer à leur pullulation en détruisant tous ceux qu'on peut atteindre. Et ce, non seulement sur les animaux eux-mêmes, par des soins hygiéniques et des agents médicamenteux, mais dans tous les points où ils auront pu parvenir. On voit qu'il s'agit surtout d'indications ayant pour base l'hygiène des locaux et celle des individus. Il serait facile, évidemment, d'étendre ces observations générales, mais les points qu'elles visent sont tellement clairs qu'il nous paraît inutile d'insister.

Aussi bien, tenons-nous à faire remarquer que la classification que nous avons adoptée dans ce rapport a été établie en vue du but spécial que nous poursuivions, à savoir la détermination des éléments prophylactiques auxquels l'homme peut avoir recours pour éviter d'être envahi par les parasites des animaux.

Nous avons tout d'abord divisé les parasites en externes et internes, et les premiers en temporaires et stationnaires.

Les parasites externes *temporaires* attaquent l'homme dans les mêmes conditions que les animaux, et méritent à peine, comme on l'a vu, d'être classés parmi les parasites ; on n'a d'autre moyen de s'en préserver que d'éviter les endroits qu'ils fréquentent de préférence, ou l'approche des animaux qu'ils recherchent plus spécialement, voire même des locaux habités par ces animaux.

Les mêmes indications s'appliquent aux parasites *stationnaires périodiques*, car si ces parasites sont toujours plus abondants dans les endroits que fréquentent ou qu'habitent leurs hôtes habituels, il est assez rare qu'ils passent directement des animaux à l'homme.

En ce qui concerne, au contraire, les parasites externes *permanents*, c'est la transmission directe, immédiate, qui prédomine de beaucoup, et il y a lieu de prendre à leur endroit des mesures de préservation plus sérieuses. Il importe, nous l'avons dit, d'éviter le contact de tous les animaux galeux dont la gale est transmissible à l'homme ; il faut recommander aux individus chargés de les soigner et de les panser, de prendre toutes les précautions voulues, et à la rigueur leur appliquer un traitement préventif ; il faut enfin désinfecter avec soin les harnais, couvertures, manteaux, etc., qui ont été en contact avec les animaux atteints et dont les hommes sont exposés à se servir. Quant aux mesures de police sanitaire, qui varient suivant les pays, elles sont d'ordre général et ne peuvent naturellement viser en particulier la transmission de la maladie à l'homme.

La prophylaxie relative aux parasites *internes* doit se régler aussi d'après la considération de leur mode habituel de transmission. Pour ceux, en effet, qui passent des animaux à l'homme par voie de transmission *médiate*, c'est presque exclusivement l'eau qui sert de véhicule, de

moyen de propagation. On arrive donc, à l'égard des parasites ordinaires comme à l'égard des microbes, à cette conclusion que l'examen des eaux potables est d'un intérêt primordial, et qu'il est indispensable d'en faire un examen attentif avant de la livrer à la consommation. Les règles qu'il convient de suivre à cet égard ont été trop souvent formulées pour que nous croyions devoir les reproduire ici. Nous nous bornerons à rappeler qu'en dehors des eaux de source, il est presque toujours nécessaire, pour éviter l'invasion des parasites, de recourir à l'ébullition ou à la filtration, parfois même à l'une et à l'autre.

Quelques parasites sont introduits dans l'organisme de l'homme à la faveur des aliments végétaux, lorsque ceux-ci sont ingérés crus ; nous avons dit suffisamment quelles précautions il convient de prendre à l'endroit de ces aliments. Il en est de même pour ceux des parasites internes qui sont transmis par les animaux eux-mêmes, avec lesquels l'homme entretient une trop grande intimité ; ce que nous en avons dit suffit à montrer la nécessité de se soustraire à cette intimité.

Le dernier cas que nous ayons eu à examiner est celui des parasites passant à l'homme par transmission *immédiate* ; à leur endroit, nous avons pu résumer les indications de la prophylaxie en ces deux formules : éviter la propagation du parasite chez les animaux, d'une part, et de l'autre empêcher l'introduction dans l'organisme de l'homme des parasites des animaux capables de s'y développer. Si la fréquence du parasitisme diminue en effet chez les animaux, l'homme court beaucoup moins de chances de se trouver infesté, et si l'homme lui-même se garantit contre l'invasion des parasites, il restreint d'autant la pullulation de ceux-ci chez les animaux. Or, les mesures à prendre pour éviter l'infestation des animaux sont avant tout sous la dépendance des règles de l'hygiène ; il s'agit essentiellement de rechercher la propreté des individus, des locaux, des aliments et des boissons. Et pour garantir l'homme lui-même, il faut lui faire connaître la nécessité de soumettre à une cuisson suffisante les viandes qui peuvent être suspectes à quelque degré, en insistant sur les caractères pratiques propres à faire constater que leur innocuité est assurée. C'est par des conférences et des livres populaires, par l'enseignement des écoles primaires, par la diffusion des notions d'hygiène en un mot, que ce résultat pourra être obtenu.

Enfin, il ne faut pas oublier que, dans une certaine mesure, l'intervention administrative peut et doit concourir à restreindre les dommages causés par les affections parasitaires. L'inspection des animaux, vivants ou abattus, dans les marchés ou dans les abattoirs, permettant de saisir et d'enlever de la consommation les viandes et les issues plus ou moins altérées par ces affections, supprime directement un nombre considérable de parasites qui eussent pu se développer dans l'organisme de l'homme et y continuer leur évolution. Et le même résultat est évidemment obtenu par les mesures de police sanitaire sagement prises et rigoureusement exécutées.

## Sur les Végétaux parasites (non-microbiens) transmissibles des Animaux a l'Homme, et réciproquement.

PAR

le Dr. Raphaël Blanchard, Professeur agrégé à la Faculté de Médecine de Paris, Secrétaire Général de la Société Zoologique de France.

Parmi les champignons parasites de l'homme, il en est quatre seulement dont on puisse démontrer sûrement la transmission directe à notre espèce par les animaux avec lesquels nous sommes ordinairement en rapport. Ce sont :—

*Achorion Quinckeanum*, Zopf, 1890 ;
*Achorion Schoenleini*, Remak, 1848 ;
*Trichophyton epilans*, Mégnin, 1878 ;
*Trichophyton tonsurans*, Malmsten, 1848.

La contagion directe des animaux à l'homme est très probable, mais insuffisamment démontrée pour les quatre microphytes suivants :—

*Actinomyces bovis*, Harz, 1877 ;
*Microsporon Audouini*, Gruby, 1843 ;
*Lepocolla repens*, Eklund, 1883 ;
*Aspergillus fumigatus*, Fresenius ;

Nous allons résumer brièvement les faits qui démontrent l'exactitude des prémisses ci-dessus énoncées.

### Achorion Quinckeanum, Zopf, 1890.

Synonymie : *Achorion Arloingi*, Busquet, 1891.

Ce champignon est la cause de la teigne faveuse de la souris. Dès 1854, Draper, médecin à New York, indiquait la transmission du favus de la souris au chat, puis de celui-ci à l'homme. Depuis lors, le professeur Saint-Cyr, de l'Ecole vétérinaire de Lyon, a apporté de nombreuses preuves à l'appui de cette même idée. C'est d'ailleurs l'Ecole dermatologique de Lyon qui, par une remarquable série d'observations, a mis hors de doute ce fait actuellement admis par tous les médecins, qu'une certaine forme de teigne faveuse nous est transmise par les petits rongeurs. La spécificité de ce favus est démontrée par deux ordres de phénomènes :—

1°. Ensemencé sur la gélatine ou dans différents milieux nutritifs, l'*Achorion Quinckeanum* donne une culture luxuriante, très différente de celle qu'on obtient avec le favus ordinaire de l'homme. C'est lui que Quincke a désigné sous le nom de microphyte *a* et que Bœr en 1887 et Busquet en 1890 ont retourné par la culture directe du favus de la souris.

2°. Au point de vue clinique, le favus provenant de la souris se distingue par des caractères très spéciaux, notamment par la rareté des godets faviques. Quincke désigne cette variété de teigne faveuse sous le nom de *Favus vulgaris*.

Le premier cas de favus chez les animaux a été observé par Jaquetant, en 1847 : deux chats de l'Antiquaille, à Lyon, étaient devenus faveux au contact de deux fillettes qui avaient l'habitude de jouer avec eux.

En 1877, Saint-Cyr a vu plusieurs élèves de l'Ecole vétérinaire de Lyon qui étaient atteints de favus : dans le placard renfermant leur linge de corps, on trouva des souris faveuses, qui avaient sans doute eté le point de départ de la contagion. Tripier s'est inoculé avec succès le favus de la souris.

D'autre part, des inoculations positives de favus ont été faites de l'homme au chat par Saint-Cyr et Vincens, de l'homme à la souris par Tripier et Vincens, et de l'homme au rat (1) par Gigard. Deux tentatives d'inoculations de l'homme au chien, faites pas Vincens, n'ont donné aucun résultat. Rien ne prouve, dans aucun de ces cas, que le microphyte transmis ait été l'*Achorion Quinckeannm* plutôt que l'*Achorion Schoenleini.*

### Achorion Schoenleini, Remak.

Ce champignon correspond aux formes $\beta$ et $\gamma$ distinguées par Quincke ; c'est lui seul que Fabry et Elsenberg ont obtenu dans leurs cultures. Il détermine cette variété de teigne faveuse que Quincke désigne sous le nom de *Favus herpeticus* et qui peut d'ailleurs s'observer parfois chez un malade, en même temps que le *Favus vulgaris.*

On admet généralement que le favus se communiqne assez rarement de l'homme à l'homme ; Alibert niait même sa contagion, mais Jaquetant, Remak et Deffis ont prouvé par des expériences rigoureuses la réalité de celle-ci. Lailler a traité à l'hôpital Saint-Louis un malade qui avait contracté la maladie en couchant dans un lit occupé précédemment par une personne atteinte de favus.

On a cru longtemps que le favus était ordinairement transmis à l'homme par le chat, contaminé lui-même préalablement par le rat ou la souris. La distinction récemment établie entre l'*Achorion Quinckeanum* et l'*Achorion Schoenleini* démontre que le *Favus herpeticus* ne nous est pas transmis par le chat, voire même par le chien, ou par les petits rongeurs, mais rend d'autre part très incertaine la provenance de cette dermatose. Assurément, la possibilité de la contagion réciproque dans l'espèce humaine rend compte de certains cas, mais ne saurait les expliquer tous. Il est probable que l'homme peut s'infester au contact de certains animaux, mais on ne saurait dire actuellement quels animaux doivent être incriminés.

Le chien est parfois atteint de teigne faveuse, mais les résultats positifs obtenus par Saint-Cyr en inoculant à cet animal le favus du chat, tendent à faire admettre qu'il s'agit habituellement de la teigne

---

(1) Dans le *Roman du Renard,* qui date du XI^e^ siècle, le rat est appelé *Pelé.* Ce nom vient-il de ce que la queue du rat est à peu près glabre, ou n'est-il pas dû plutôt à ce que, à cette époque, le rat (*Mus rattus*) était déjà frèquemment atteint de tèigne faveuse ?

causée par l'*Achorion Quinckeanum*. Par ses rapports journaliers avec le chat familier, le chien doit d'ailleurs se contaminer assez aisément. Le chien contracte aussi sans difficulté la teigne du lapin, d'après d'autres expériences de Saint-Cyr.

Le cheval et le bœuf peuvent également être atteints de favus, encore que ce soit là une manifestation morbide assez rare. En 1880, Gigard a observé une épidémie de teigne faveuse qui sévissait tout-à-la fois dans l'espèce bovine et chez les enfants : dans ce cas, la transmission de l'animal à l'enfant ne semblait pas douteuse. Il y a là une indication précieuse, au point de vue de l'étiologie du favus humain.

Ercolani décrit en 1876, sous le nom d'*Achorion keratophagum*, un microphyte qu'il a rencontré dans le sabot des solipèdes atteints de fourmilière et qu'il considère comme la cause de cette maladie. En raison de l'analogie de celle-ci avec la rogne ou carie sèche des ongles humains, Ercolani admet encore que la rogne est causée par le même parasite. Cette opinion ne repose d'ailleurs sur aucune observation positive, et la spécificité de l'*Achorion keratophagum* n'est pas plus démontrée au point de vue botanique qu'au point de vue clinique.

En 1858, Müller, Gerlach et d'autres ont observé chez le coq et la poule un favus de la crête et des caroncules. En 1881, Mégnin étudie avec soin le champignon qui cause cette affection et lui donne le nom d'*Epidermophyton gallinæ*, le considérant comme spécifiquement distinct de l'*Achorion Schoenleini*. Les parties malades présentent des croûtes blanches, farineuses ou plâtreuses ; les godets faviques font défaut. Le microphyte est caractérisé par un mycélium fin, court, tortueux, émettant des sporophores cloisonnés, terminés par des chapelets de 5 à 6 spores rondes, larges de 6 à 8 $\mu$, plus volumineuses que celles de tous les autres champignons parasites de nos animaux domestiques. Il pullule entre les lames épidermiques, sans s'introduire dans les follicules plumeux. Il se cultive bien sur la gélatine ; il s'y développe à la surface en touffes d'un blanc de neige, tandis que la gélatine se liquéfie et prend la couleur du jus de groseille.

Au contraire, Neumann (de Toulouse) assure que le favus des poules est dû simplement à l'*Achorion Schoenleini*. Il base son opinion sur la ressemblance morphologique des deux champignons et sur le résultat de quelques inoculations. Chez un jeune chien, inoculé avec le favus de la poule, il se développe une dermatose identique à celle qu'on obtient chez un autre chien, après inoculation du favus de l'homme. Cette même expérience comparative réussit sur deux lapins, sans que rien permette de soupçonner l'origine différente des deux affections. Inversement, on reproduit le favus de la poule en déposant sur la crête des croûtes de favus humain délayées dans l'eau. De ces expériences il semble donc résulter que la poule doit être envisagée comme l'une des sources du favus humain.

### Trichophyton Depilans, Mégnin, 1878.

Ce microphyte est la cause de la teigne tonsurante dans l'espèce bovine. Ernst, médecin dans le canton de Zurich, signalait dès 1820 que l'herpès tonsurant peut être communiqué à l'homme par les

animaux : une jeune fille avait manifestement contracté la maladie en trayant une vache qui en était elle-même atteinte. Des faits de ce genre ont été signalés à l'attention des médecins par Grognier en 1831, Kollreuter en 1836, Lavergne et Fehr en 1838, Epple en 1839, Rademacher en 1842, Houlez (de Sorèze) en 1845, Horing en 1846, Letenneur et Malherbe (de Nantes) en 1851, puis par Reynal en 1858.

La maladie acquise ainsi par l'homme est un herpès circiné qui diffère notablement de l'herpès tonsurant ordinaire. Mégnin a complété ces démonstrations en mettent en relief, en 1878, les différences essentielles, tant morphologiques que cliniques, qui caractérisent le *Trichophyton depilans*.

A l'aide d'expériences directes, Gerlach a démontré que la teigne tondante est transmissible du bœuf au bœuf, du bœuf au cheval, et du bœuf au chien ; toutefois, chez ce dernier, l'inoculation réussit assez difficilement. Gerlach n'a jamais eu qu'un résultat négatif en cherchant à contaminer le mouton et le porc par le bœuf, mais Perroncito a observé la transmission de la teigne du bœuf à l'agneau.

TRICHOPHYTON TONSURANS, Malmstén, 1848.

En 1853, Bazin a publié la fameuse observation de gendarmes qui avaient contracté la teigne tondante en soignant des chevaux atteints de "dartre." Galligo en 1858, Horand (de Lyon) en 1871, Dieu en 1876, Larger en 1881, et Longuet en 1882 ont publié des cas analogues.

En 1881, Mégnin a observé une quinzaine d'artilleurs d'une même batterie, qui présentaient des cercles d'herpès circiné sur le cou et le menton. Etant au camp pour les manœuvres, ils avaient pris les couvertures des leurs chevaux pour se préserver du froid et s'y étaient enveloppés jusqu'au menton. Or, ces chevaux étaient récemment arrivés des dépôts de remonte et étaient atteints de dartre tonsurante, comme un examen attentif permit de le constater.

Le cheval peut donc transmettre à l'homme la teigne tonsurante. Cette transmission s'observe même assez fréquemment dans les régiments de cavalerie. En France, la maladie est importée par de jeunes chevaux venus des haras de Normandie, pays où la teigne tonsurante existe à l'état endémique chez les animaux d'espèce bovine. Si, comme on l'a dit, les poulains se contaminent dans les pâturages, au contact des bœufs et des veaux, ou bien la teigne tondante du cheval pourrait reconnaître une double origine (*Trichophyton depilans* et *Tr. tonsurans*, suivant les cas), ou bien le *Trichophyton depilans* serait une simple variété du *Trichophyton tonsurans*. C'est là une question importante, sur laquelle il est difficile de se prononcer actuellement.

La teigne tonsurante peut aussi nous être communiquée par le chien ; les cas étudiés par Purser en 1865 et par Horand en 1872 et en 1873 en donnent une preuve convaincante. L'observation de Purser et la première observation de Horand sont particulièrement remarquables, en ce qu'elles nous montrent que l'onychomycose ou trichophytie unguéale peut provenir du chien.

Le chat lui-même peut devenir un agent de transmission. Tuckwell en 1871, Lancereaux et Michelson en 1874, ont publié des observations qui ne laissent aucun doute à cet égard.

Quant à la transmission de la teigne tondante des ruminants à l'homme, on doit faire des réserves expresses, car nous pensons que, dans la majorité des cas, sinon dans tous, la contagion s'est faite par l'intermédiaire du *Tricophyton depilans*.

Toutefois, nous ne pouvons affirmer que ce champignon soit la cause exclusive de la trichophytie bovine, puisque le *Trichophyton tonsurans* s'inocule très facilement au veau. La teigne tondante de l'homme se transmet aussi très aisément au chien (Cramoisy, 1856; Vincens, 1874) et au chat (Vincens), mais ne se transmet pas aux rongeurs (souris, rat, lapin). Ajoutons que cette même dermatose se propage tout aussi bien entre animaux d'espèce différente, par exemple du chat au cheval (Williams).

Mégnin a décrit chez le lapin, sous le nom de *Teigne lycoperdoïde*, une maladie que certains auteurs considérent comme identique à la trichophytie tonsurante, mais qui semble bien en être distincte, d'abord à cause de ses caractères cliniques très spéciaux, puis à cause de la difficulté (pour ne pas dire l'impossibilité) que l'on éprouve à transmettre au lapin la teigne tondante de l'homme.

### Actinomyces Bovis, Harz, 1877.

Ce champignon ne saurait rentrer dans le genre *Actinomyces*, Meyen, 1827; ce serait donc un acte de justice que de le désigner sous le nom de *Discomyces*, proposé par Rivolta. La maladie qu'il détermine et qui porte le nom d'*actinomycose*, prendrait alors celui de *discomycose*.

En raison même du titre de ce rapport, il peut paraître hors de propos de mentionner ici l'*Actinomyces*, que certains auteurs rangent parmi les Desmobactériacées, à côté du *Cladothrix dichotoma*. Nous ne méconnaissons point la valeur des raisons invoquées en faveur de cette opinion. Si pourtant nous continuons à rattacher l'*Actinomyces* aux champignons proprement dits, c'est uniquement parce que ses caractères morphologiques ne nous semblent pas suffisamment établis pour qu'on puisse lui attribuer une place certaine et définitive dans la classification.

L'actinomycose n'a encore été observée que chez l'homme, le bœuf, le cheval et le porc; chez les trois premiers, elle siège dans des organes très différents, mais surtout au voisinage du tube digestif ou dans le poumon, d'où l'on peut conclure que le champignon pathogène s'introduit dans l'organisme soit avec les aliments, soit avec l'air inspiré. Chez le porc, on ne le trouve que dans les muscles. En somme, et c'est là un fait capital, l'actinomycose n'est pas une maladie de la peau ou des muqueuses, ce qui constitue déjà un premier argument contre la nature contagieuse de cette affection.

Elle ne semble pas davantage être transmissible par l'usage de la viande malade. D'ailleurs les carnivores, non seulement ne contractent pas spontanément la maladie, mais encore ne la prennent pas par

inoculation directe des cultures pures d'*Actinomyces*; en revanche, l'inoculation réussit très bien chez le lapin.

La contagiosité de l'actinomycose est admise par divers auteurs : Hacker, Stelzner et Israël ont cité des cas où des personnes auraient été contaminées parce qu'elles se trouvaient en contact avec des animaux malades. En 1888, Boracz a publié l'observation d'un cocher qui, sans jamais avoir été en rapport avec des animaux malades, était pourtant atteint d'actinomycose du maxillaire; quatre mois plus tard, sa femme présentait elle-même tous les signes de la maladie.

En se basant sur ces observations, on admet la contagion de l'animal à l'homme et même de l'homme à l'homme. Cette conclusion est-elle rigoureuse? Nous ne le croyons pas.

Les observations ci-dessus démontrent simplement que les individus qui en sont l'objet se sont trouvés dans les conditions mêmes dans lesquelles le bétail contracte la maladie. Or, ces conditions sont actuellement connues : on soit que l'*Actinomyces* se trouve répandu sur les graminées et qu'il est introduit dans le poumon par la poussière émanant de celles-ci ou sous la peau et les muqueuses par les barbes de céréales. N'est-il pas vraisemblable que les deux individus dont il est question dans l'observation de Boracz ont pu être contagionnés de cette manière? Et la même explication n'est-elle pas également valable pour les bouviers, gens de ferme, etc., qui contractent la maladie en même temps que le bétail et dans des conditions sensiblement identiques?

Nous trouvons un dernier argument contre la contagiosité dans les statistiques publiées par divers auteurs, notamment par Moosbrügger, qui a relevé jusqu'à 75 cas. Sur ce nombre, 10 cas seulement se rapportaient à des propriétaires fonciers, à des paysans, à des valets de ferme; tous les autres cas s'observaient chez des personnes appartenant aux professions les plus diverses et pour lesquelles la contagion directe ne saurait être admise.(1)

### Microsporon Audouini, Gruby, 1843.

Synonymie : *Microsporon decalvans*, Bazin, 1853. *Trichophyton decalvans*, Bazin, 1873.

On peut distinguer trois formes de pelade :—

1°. Une pelade non parasitaire et non contagieuse, due vraisemblablement à une trophonévrose.

2°. Une pelade parasitaire, due à un *Micrococcus* qui envahit le follicule pileux. Cette pelade, entrevue par Thin en 1881 et par von Sehlen en 1883, a été bien étudiée récemment par H. Nimier, puis par L. Vaillard et H. Vincent; elle est probablement contagieuse, mais rien ne permet encore de supposer qu'elle ait le moindre rapport avec une maladie quelconque des animaux.

---

(1) Au moment où nous corrigeons les épreuves de cet article, nous pouvons prendre connaissance des comptes-rendus sommaires du Congrès d'hygiène, publiés par les journaux de médecine. Nous y voyons que M. Crookshank (de Londres) et M. le professeur Nocard (d'Alfort) ont soutenu, relativement à l'origine de l'actinomycose, une opinion toute semblable à celle que nous exposons plus haut.

3°. Une pelade parasitaire, due au *Microsporon Audouini* et se présentant sous deux aspects : pelade achromateuse et pelade décalvante. Bazin considérait cette dernière comme une entité morbide distincte et lui attribuait un microphyte spécifique.

La pelade par *Microsporon*, la seule qui doive nous arrêter, est transmissible de l'homme à l'homme : le fait est suffisamment démontré pour qu'il soit inutile d'en donner des preuves nouvelles. En revanche, la transmission à l'homme de l'alopécie des animaux, bien que vraisemblabe, n'est nullement certaine.

Dès 1856, Rivolta (1) a observé un bœuf qui portait sur le ventre une plaque d'alopécie : " peu-à-peu elle s'étendit et devint presque " générale ; tous les poils tombèrent et la peau resta d'une couleur " obscure brillante. Les paysans croyaient cette maladie contagieuse et " la craignaient." En 1874, Hillairet a publié la curieuse observation de six employés du chemin de fer de l'Est, à Paris, tous atteints de pelade et auxquels, selon toute vraisemblance, la maladie avait été communiquée par un chat. Arnozan (de Bordeaux) a fait connaître en 1885 une série de cinq observations de pelade chez des personnes qui avaient été probablement contaminées par des animaux (chien et chat) atteints eux-mêmes d'alopécie ; la recherche du *Microsporon* spécifique n'a été fait dans aucun de ces cas.

Il est certain du moins que la pelade est fréquente chez le jeune chat, comme l'a fait remarquer Baillet, et que sa propagation de chat à chat est incontestable. L'alopécie circonscrite est également assez commune chez le chien (Siedamgrotzky), chez le veau (Perroncito) et se voit même à la racine de la queue du cheval ; dans ce dernier cas, suivant Leisering, l'" *herpes caudalis* " est caractérisé par la présence de champignons.

Si des observations cliniques ultérieures viennent confirmer les faits qui précèdent ; si d'autre part des inoculations de l'animal à l'homme et de l'homme à l'animal démontrent la transmissibilité de la pelade ; si enfin les cultures des champignons recueillis tant sur l'homme que chez les animaux se montrent toujours identiques, l'exactitude des prévisions que nous venons d'énoncer sera définitivement établie. A ces conditions seulement il sera possible d'affirmer que la pelade de l'homme et l'alopécie des animaux sont une seule et même maladie. En attendant cette démonstration rigoureuse, il sera prudent de tenir à l'écart et de traiter par les procédés antiparasitaires tout animal dont les poils tombent par plaques.

### Lepocolla repens, Eklund, 1883.

Synonymie : *Epidermophyton*, Lang, 1879 (nec Mégnin, 1881).

Pour certains auteurs, le psoriasis est une affection d'origine purement nerveuse, non parasitaire et non contagieuse ; pour d'autres, c'est, au contraire, une dermatomycose.

---

(1) *Dei parassiti vegetali*, p. 469. Torino, 1873.

Dès 1878, Lang (d'Insbrück) a soutenu cettee dernière opinion. Dans les squames psoriasiques, il trouve un champignon qu'il désigne sous le nom d'*Epidermophyton* et dont la présence est constante : s'il est resté jusqu'alors inaperçu, cela s'explique bien plus par la pâleur de ses filaments mycéliens et de ses spores que par leur exigüité. Balzer (de Paris) en 1881 et Eklund (de Stockholm) en 1883 retrouvent le même parasite.

Ce dernier observateur croit reconnaître que la maladie débute par les capillaires des papilles dermiques : le mycélium se développe autour de ces capillaires, puis envahit la profondeur de la couche muqueuse de Malpighi, dont les cellules se montrent bientôt enserrées dans un lacis de filaments mycéliens, portant des spores en massue. Les écailles de psoriasis, ensemencées dans des milieux convenables, donnent un abondant mycélium, dans les filaments duquel se forment des spores endogènes. La démonstration eût été convaincante, si la maladie avait été reproduite expérimentalement, par inoculation des cultures ; mais cette recherche n'a pas été faite.

Wolff (de Strasbourg) a observé, lui aussi, le *Lepocolla ;* les spores siégeraient de préférence dans les parties les plus profondes de l'épiderme.

Quelques auteurs, tout en admettant la nature parasitaire du psoriasis, ne croient pas à la spécificité du *Lepocolla :* telle est du moins l'opinion d'Unna et de Quinquaud. Sans se prononcer sur la question, Mapother déclare simplement que l'agent pathogène est un organisme anaérobie, qui ne vient pas directement du dehors, mais envahit plutôt la peau par les capillaires du derme, qu'il obstrue et qu'il finit par déchirer.

En somme, la théorie parasitaire du psoriasis compte des partisans sérieux et convaincus.

La contagion de la maladie dans l'espèce humaine a d'ailleurs été mise en évidence par Unna (de Hambourg), qui vit une domestique psoriasique, récemment admise dans une famille, transmettre le psoriasis à trois enfants confiés à sa garde. Aubert (de Lyon) a vu, de son côté, un mari transmettre la maladie à sa femme. Enfin, Augagneur (de Lyon) a observé à l'Antiquaille un teinturier chez lequel se déclara un psoriasis progressivement généralisé, au bout de quatre mois de séjour à l'hôpital, entre deux psoriasiques ; l'intérêt très spécial de cette observation réside en ce que ce malade était primitivement atteint d'un eczéma professionnel, qui se transforma directement en psoriasis.

D'après ces faits, on peut donc penser que les cas que, jusqu'à présent, on a cru pouvoir attribuer à l'hérédité, s'expliquent plus justement par une transmission direction des parents aux enfants.

Le psoriasis humain est contagieux pour les animaux. A la suite de frictions répétées avec des squames recueillies sur un malade, Lassar a pu inoculer trois lapins. Des expériences analogues, variées de diverses façons, ont été faites par Tommasoli et par Beissel. Non seulement la maladie se développe chez le lapin, quand on frictionne la peau de ce rongeur, mais même quand on lui injecte sous la peau, dans

le péritoine ou dans la veine jugulaire, des squames psoriasiques délayées dans une solution faible de chlorure de sodium. Bien plus, le psoriasis communiqué expérimentalement par le lapin est transmissible à un autre lapin, auquel cas la période d'incubation est remarquablement courte.

A côté de ce psoriasis expérimental et artificiel, il convient de rappeler qu'une affection psoriasique, peut-être identique au psoriasis humain, s'observe chez le cheval, l'âne et le mulet, ce qui, selon la juste remarque d'Eklund, explique pourquoi les cochers sont assez fréquemment atteints par la maladie.

Un psoriasis inoculable à l'homme frappe également l'espèce bovine, et c'est là, pensons-nous, un fait d'une importance capitale, car une étude attentive pourrait montrer la fréquence des faits dont nous allons parler et jeter une vive lumière sur l'étiologie toujours obscure du psoriasis humain.

En 1887, Tenholt constata que des bœufs d'origine hollandaise étaient atteints d'une maladie cutanée qui avait la plus grande ressemblance avec le psoriasis. Elle se communiquait de l'animal à l'animal et passa aussi sur quatre individus chargés de garder et de soigner les animaux malades. Des cultures pures et l'inoculation de celles-ci sur des bœufs sains ne donnèrent aucun résultat positif. Il semble néanmoins démontré que l'homme puisse acquérir le psoriasis au contact du bœuf.

L'origine bovine de la maladie est, d'autre part, mise hors de doute par une importante série d'observations dans lesquelles on a vu nettement la maladie débuter, chez l'enfant et chez l'adulte, au niveau d'une pustule vaccinale obtenue avec le vaccin de génisse. Les premiers cas de ce genre ont été constatés aux Etats-Unis, en 1883, par Piffard, Th. Wood, Biart et Rohé; des observations identiques ont bientôt été faites en France, à Lyon, par Chambard, puis par Augagneur.

### Aspergillus fumigatus, Fresenius.

Dieulafoy, Chantemesse et Widal [1] ont reconnu, en 1890, chez les pigeons vendus sur les marchés de Paris, une affection qui détermine, à la surface de la muqueuse buccale et dans le poumon, des lésions tout à fait comparables à celles de la tuberculose bacillaire, mais qui pourtant n'est point due au bacille de Koch. Les tumeurs contiennent à leur centre un mycélium qui, cultivé par les méthodes usuelles, acquiert tous les caractères de l'*Aspergillus fumigatus*. L'inoculation des spores à des pigeons détermine plus ou moins rapidement des lésions pseudo-tuberculeuses identiques à celles qui se développent spontanément chez ces animaux.

Bien qu'aucune autopsie ne soit encore venue en donner une preuve indiscutable, on peut affirmer que la même pseudo-tuberculose s'observe à Paris, chez les gaveurs de pigeons. Leurs expectorations ne renferment pas de bacilles, mais bien des fragments mycéliens. L'inoculation

---

(1) Dieulafoy, Chantemesse et Widal, *Une pseudo-tuberculose mycosique* Gazette des hôpitaux LXIII., No. 89, p. 821, 1890.

d'un crachat de malade à un pigeon produit chez ce dernier une tuberculose mycosique due à l'*Aspergillus fumigatus ;* l'ensemencement des crachats sur la gélose donne des colonies de ce même *Aspergillus.*

Voilà donc une aspergillose qui s'observe tout à la fois chez l'homme et chez l'oiseau. Existe-t-il une relation entre ces deux êtres, au point de vue de la propagation de la maladie?

Pour pratiquer le gavage, le gaveur se remplit la bouche d'un mélange d'eau et de graines, puis ouvrant le bec du pigeon, il y applique ses lèvres et chasse par expiration une partie du mélange. Il est vraisemblable que le gaveur prend l'*Aspergillus,* cause de la pseudo-tuberculose pulmonaire, soit à la surface des graines qu'il introduit dans sa bouche, soit au contact direct de la tumeur buccale du pigeon. Il est même possible que celui-ci soit contaminé par l'homme qui, en soufflant pour projeter les graines, introduit dans les voies aériennes de l'oiseau quelques germes d'*Aspergillus.*

### Microphytes insuffisamment connus.

En outre des champignons étudiés ci-dessus et dont les rapports avec les maladies contagieuses sont définis plus ou moins nettement, on connaît chez les vertébrés allantoïdiens un certain nombre de microphytes qui causent ou du moins accompagnent des maladies cutanées, mais dont on ignore encore l'origine ou les relations avec les maladies des autres animaux. Nous croyons utile d'en dresser la liste et d'indiquer très succinctement les affections dans lesquelles on les observe et les lacunes que présente leur histoire, dans le but d'attirer sur eux l'attention et de susciter des recherches qui puissent nous renseigner exactement sur leur valeur pathogénique, sur leur provenance et sur le degré de leur transmissibilité.

1°. *Selenosporium cuticola*, R. Bl., 1891.—Nous désignerons désormais sous ce nom un champignon dont nous avons fait un étude détaillée et qui cause, chez le lézard vert, une remarquable dermatose, probablement contagieuse pour les sauriens.

2°. *Microsporon pterophyton*, Mégnin, 1878.—Sur deux cacatoès qui perdaient leur plumage, Mégnin a reconnu que les plumes "étaient " envahies par un *Microsporon* très petit, englobant les barbules d'un " véritable feutre. Le mycélium est très distinct. Les sporules ont 1 à " 2$\mu$." Mégnin ne dit pas avoir constaté la présence du microphyte dans la peau; il n'est donc pas certain que celui-ci soit la cause de la chute des plumes. D'autre part, l'existence d'un mycélium donne à penser qu'il n'appartient pas au genre *Microsporon.*

3°. *Botryomyces.*—Ce champignon a des affinités manifestes avec l'*Actinomyces.* Signalé d'abord chez le cheval, il a été vu par Csokor (de Vienne) sur la mamelle de la vache. On ne sait rien de sa provenance ni de sa contagiosité.

Les champignons suivants sont particuliers à l'espèce humaine :—

4°. *Chioniphe Carteri.*—Il a été observé dans les cas de mycétôme ou pied de Madura. Sous ce nom, les auteurs ont décrit des maladies évidemment très diverses; certaines observations semblent se rapporter simplement à l'actinomycose.

5°. *Oïdium albicans*, Ch. Robin.—Ce champignon est bien connu au point de vue clinique ; Linossier et Roux (de Lyon) l'ont étudié avec le plus grand soin au point de vue biologique. Néanmoins, on ignore encore s'il provient exclusivement de germes propagés par l'air ou s'il ne serait pas, dans certains cas, amené dans la bouche des enfants avec le lait de vache.

6°. *Oïdium lactis*.—On l'a vu dans le lait de brebis ; peut-être provoque-t-il des accidents chez de jeunes nourrissons ou chez de jeunes animaux nourris avec du lait. Ses relations avec l'*Oïdium albicans* méritent d'être déterminées avec plus de précision.

7°. *Microsporon anomæon*, Vidal, 1883.—Sous le nom de *Microsporon anomæon* ou *dispar*, le savant médecin de l'hôpital Saint-Louis a fait connaître un microphyte de l'épiderme, qu'il considère comme la cause du pityriasis circiné et marginé. En 1887, Mannino croit retrouver ce même parasite dans la séborrhée et lui conteste tout rôle pathogénique et spécifique ; nous pensons que le médecin italien a plutôt eu affaire au *Microsporon ovale*.

8°. *Microsporon furfur*, Ch. Robin, 1853 (*Malassezia furfur*, H. Baillon, 1889).—Le pityriasis versicolor est contagieux dans l'espèce humaine ; on ne sait rien de ses relations avec les maladies analogues chez les animaux.

9°. *Microsporon minutissimum*, von Bärensprung, 1862 (*Microsporon gracile*, Balzer, 1883).—Il cause l'érythrasma, affection dans laquelle Burchardt l'a découvert en 1859.

10°. *Microsporon ovale*, Bizzozero, 1884 (*Saccharomyces ovalis*, Bizzozero, 1884 ; *S. sphaericus*, Bizzozero, 1884, nec Nägeli, 1879 ; *S. capillitii*, Oudemans et Pekelharing, 1885 ; *Cercosphaera capillitii*, Oudemans et Pekelharing ; *Microsporon Malassezi*, H. Baillon, 1889).—Nous croyons pouvoir établir la synonymie ci-dessus pour l'organisme que Malassez (de Paris) a découvert en 1874 dans le pityriasis simplex.

11°. *Microsporon trachomatosum*, Noiszewski, 1890.—Sous le nom de *Microsporon trachomatosum sive jagium*, Noiszewski décrit un microphyte auquel il attribue la production du trachôme ou conjonctivite granuleuse à forme chronique. Par la culture pure de fragments d'une cornée malade, on obtient un mycélium.

12°. *Trichophyton ovoïdes*, Behrend, 1890.—Il cause la piédre ou trichomycose nodulaire et vit sur les cheveux ; en culture pure, il se développe en un mycélium.

13°. *Monilia sputicola*, Galippe, 1885.—Il a été trouvé dans la salive humaine ; il y passe inaperçu et y végète mal, se reproduisant sans doute uniquement par bourgeonnement des spores. Cultivé, il donne un riche mycélium dont les hyphes portent à leur extrémité des chapelets de spores.

## Conclusions.

Les brillants travaux de Bazin et de ses élèves (école de Paris), puis d'Hebra et de ses élèves (école de Vienne) avaient fini par apporter

assez de lumière dans l'étude difficile des dermatomycoses, pour qu'on pût croire définitivement acquise cette notion, que les champignons de la peau sont essentiellement au nombre de quatre, causant chacun une maladie spéciale :

1°. Le *Microsporon furfur*, causant le pityriasis versicolor ;

2°. Le *Microsporon Audouini*, causant la pelade ;

3°. L'*Achorion Schoenleini*, causant la teigne faveuse ;

4°. Le *Trichophyton tonsurans*, causant les diverses formes de trichophytie et spécialement la teigne tondante.

Les recherches récemment faites en différents pays sont venues anéantir cette croyance. Non seulement le nombre des microphytes cuticoles a augmenté dans une large mesure, mais encore on a acquis la conviction que, sous les noms de favus et de trichophytie, on avait confondu plusieurs affections bien distinctes, caractérisées chacune par un organisme parasitaire spécifique.

Ces recherches sont encore trop récentes, elles sont d'ailleurs encore trop inachevées pour qu'on puisse prévoir où l'on s'arrêtera dans cette voie nouvelle et, par conséquent, pour qu'on puisse dès maintenant en tirer des conclusions définitives. Il est acquis néanmoins que des animaux d'espèce très variée (poule, bœuf, cheval, lapin, rat, souris, chien, chat) peuvent transmettre à l'homme, soit par une fréquentation assidue, soit par un contact passager, des maladies parasitaires capables de se développer chez celui-ci, d'y devenir plus ou moins tenaces et d'y provoquer des accidents plus ou moins graves.

Ces notions, basées tout à la fois sur l'observation clinique, sur la culture des microphytes en dehors de l'organisme et sur la reproduction expérimentale de la maladie, par inoculation soit de ces cultures, soit simplement de fragments épidermiques renfermant le parasite, rendent légitimes les conclusions suivantes :—

1°. Un certain nombre de dermatoses, causées par des champignons, sont transmises à l'homme par les animaux avec lesquels celui-ci peut se trouver en contact.

2°. Par la fréquentation journalière des animaux domestiques, l'homme est donc exposé à contracter certaines maladies cutanées.

3°. Tout animal dont la peau présente un aspect anormal (desquamation, croûtes, etc.) ou dont les poils semblent tomber ou se briser spontanément, doit être isolé, soumis à une stricte observation et traité par les procédés parasiticides.

4°. La litière ayant servi à cet animal sera détruite par le feu. Les stalles d'écurie, chenils, harnais, couvertures seront désinfectés. Les tapis, couvertures de lit, etc., sur lesquels les chats et les chiens se couchent volontiers, seront passés à l'étuve.

5°. L'homme peut lui-même communiquer certaines dermatomycoses aux animaux domestiques.

6°. Tant que le traitement médical n'aura pas arrêté le mal ou du moins ne l'aura pas suffisamment atténué pour que, de l'avis du médecin, toute chance de contamination ait disparu, on devra s'abstenir de tout contact avec les animaux, ceux-ci pouvant s'infester à leur tour et devenir ainsi, secondairement, les propagateurs de la maladie.

7°. Les différents gouvernements devront prohiber, par un règlement spécial, l'introduction sur leur territoire de tout animal de provenance étrangère, chez lequel on aura notoirement reconnu l'existence d'une dermatose parasitaire et transmissible, non seulement de l'animal à l'homme, mais aussi de l'animal à l'animal.

## DISCUSSION.

**Professor Brown, C.B.,** said: that after the very elaborate papers which had been already read, and particularly the last paper, which dealt so very minutely with some of the microphytes—indeed with a large number of species which were hardly known among scientists in this country—it would not be necessary for him to go into great detail. What he proposed to do was to discuss the subject generally, and endeavour to illustrate some of the remarks of previous speakers by the aid of microscopic lantern slides projected on the screen. It had always been perfectly well recognised that there were certain organisms which maintained an existence in other organisms. This condition was known under the general name of symbiosis, which simply meant association. The condition of parasitism was capable of being looked at from more than one point of view, and it was quite certain that parasitism included very various degrees of injury. He might remark that in numerous cases where outbreaks of disease amongst lambs during the past season had been ascribed to parasites, they had been able to show by the stern logic of facts that the parasites had nothing to do with the disturbance. They had found the same parasites in other lambs which were absolutely free from disease of any kind, and in the most flourishing condition. In other cases, however, parasites did inflict an enormous amount of mischief, and this was true, not only of the animal parasites, but also of those numerous microphytes which were derived from the vegetable kingdom, and which found a habitat generally on the surface of the higher animals, though occasionally in the internal organs.

Professor Brown then proceeded to describe in detail the characteristics of a variety of parasites by lantern demonstrations. Amongst the parasites thus described, were the following:—

Cestodes { Large cysts, *Tenuicollis.*
Small cysts, *Echinococcus.*
Tape worms, *Tænia lata* (sheep).
,, ,, *Tænia serrata* (dog).

Trematodes: Fluke, egg, embryo, sporocysts, cercaria.

Nematodes: Strongles, lung of sheep.

Acanthocephala: Thorn-headed worms.

In concluding, he observed that if he attempted to explain in detail the many ways in which animal parasites could be conveyed, he should only be quoting the words of Dr. Cobbold, who was certainly the greatest helminthologist of his day, at least in this country, and who had laid the basis of the work on which Englishmen had subsequently proceeded. He would only add that whilst parasites were naturally looked upon with a sentiment of disgust, and were charged with being the cause of every conceivable form of mischief, there was a point of view from which he apprehended they might be considered as suffering from injustice in the

struggle for existence. One could understand that a parasite like that on the screen might feel itself grossly illtreated by being expelled from the intestine in which it was placidly remaining, and it might allege on its own behalf, that it had as much right to infest the intestines of animals, as those animals had to infest or to live on others. There was no doubt, he admitted, that in many cases parasites did a considerable amount of mischief, and that in other instances they inflicted fatal injury; but at the same time the argument might be turned with terrible effect upon the human beings who complained of their ravages, if the question were fairly put to them: "Are you yourself free from that propensity to invade, "not only doing considerable mischief, but inflicting fatal injury, upon "your fellow worm?"

**Professor Perroncito** (Turin) dit:—Je profite de l'occasion pour communiquer deux observations d'actinomycose de la peau qui peut-être doivent modifier les idées manifestées par mon très honoré ami et collègue Professeur Blanchard. L'une a été faite par moi avec notre très distingué M. Charles Reymond, Professeur d'Ophthalmologie à Turin; l'autre me l'a offert un Vétérinaire militaire avec M. le Dr. Perosino de Turin. Le premier cas se réfère à une dame soignée par M. Reymond. Cette dame avait à une cuisse un nodule ulcéré qui résista à tout soin. M. Reymond a exporté une petite pièce de la tumeur dans laquelle nous avons trouvé les *cespugli* de l'*actinomycès* identiques à *l'actinomycès bovis*. Le second cas se référe à un cheval de l'artillerie, qui dans les manœuvres a été blessé à une cuisse avec une pierre. Le cheval a été mis dans une étable pour être soigné. Mais la plaie n'est jamais guérie complêtement; elle s'est au contraire mieux ouverte, et après une année le cheval a dû être tué par une actinomycose de la peau qui s'est transmise jusqu'à l'os.*

Ces deux faits démontrent comme l'actinomyces peut se transmettre pour la peau. Relativement aux parasites des animaux et plus spécialement les protozoaires, pour se transmettre entre les animaux, des animaux à l'homme et *vice versâ*, ils ont beaucoup de fois besoin de passer à l'état d'enkystément à l'état libre, précisément comme la trichine passe à l'état d'encapsulément dans les muscles des animaux. J'ai démontré dans l'année 1887 pour le *megastoma intestinalis*† et dans le 1888 pour les autres flagellés parasites de l'intestin de l'homme‡ que dans des conditions tout-à-fait spéciaux, comme j'ai expliqué dans mes communications annoncées, les flagelles surdits se revêtent d'une membrane kitineuse qui serve à transformer les cercomonas dans un état de vie durable, semblable à une spore d'un bactère. Ainsi les protozoaires peuvent résister à dessication et à d'autres vicissitudes atmosphériques pour rentrer dans le corps de l'homme ou des animaux et perpétuer l'espèce. Aussi dans le rat j'ai demontré spérimentalement que la *Lamblia intestinalis* de notre intestin administrée avec l'aliment, après six jours ont trouvé déjà dans les selles les megastoma encapsulés. Ce sont probablement les rats qui servent à propager l'espèce parasitaire dans l'homme. Un encapsulément analogue§; (je l'ai décrit dans le 1880), par les larves mûres de *l'Ankylostoma duodenalis*. Alors M. Leuckart, Grassi et autres ont objecté qu'on avait à faire plutôt

---

* Voir le livre par E. Perroncito: I Parassiti dell' uomo e degli animali utili. Milano, 1882.

† E. Perroncito: L'Enkystement du *megastoma intestinalis*. R. Academia di Medicina di Torino, Maggio, 1887.

‡ Sulla diffusione del *cereomonas intestinalis*. Turin, 1888.

§ *E. Perroncito* osservazioni elmintologoche relative all' anemia soltassi epidemia vegli operai del Gottardo (R. Accademia dei Lincei—Seduta del 2 Maggio, 1880).

avec une mue. Mais j'aurai l'honneur de vous présenter des préparations que j'espère vous démontront le fait dans une manière la plus probante: c'est dans ces conditions de larves mûres à l'état libre que parmi les eaux, les aliments, la terre, et aussi pour l'air peut se dissoudre l'espèce parasitaire.

## Wednesday, 12th August, 1891.

The PRESIDENT, Sir NIGEL KINGSCOTE, K.C.B., in the Chair.

VICE-PRESIDENTS of the day:
Professor CHAUVEAU, Paris.
GEORGE BUCHANAN, M.D., F.R.S., Medical Officer of the Local Government Board, London.

## On Meat-Infections—Food Poisoning.

BY

EDWARD BALLARD, M.D., F.R.C.P., F.R.S.

The subject of the Infection of Meat, Milk, and other comestibles is one which has come largely into prominence of late years, during which the mode of morbific infection of food and the manner in which such infection comes to operate on the human body have been made matter of diligent research. Carrying back my memory to an antecedent period, little was known beyond this, that occasionally symptoms of poisoning followed the use of apparently wholesome kinds of food, but the phenomena of food-poisoning were not in any case that I recollect identified with those of any known specific disease, with the exception perhaps of choleraic diarrhœa or cholera nostras.

Milk especially, prior to about 20 years ago, was regarded as invariably a particularly wholesome and harmless kind of food; and gastro-intestinal disturbances, obviously resulting from the eating of flesh-foods, or of soups, &c., prepared from them, were commonly attributed to accidental impregnation of the food with such metallic poisons as copper or antimony, or, in rare instances, to some chemical change in its organic constituents, the nature of which, if it differed at all from ordinary putrefaction, was unexplained, and at that time inexplicable. Indeed, much of the etiological research of the last 20 years has had reference to the production of disease in man through the agency of his food. Investigations into the spread of certain specific fevers, such as enteric fever and scarlet fever, through milk, took the lead at all events in this country, investigations which have always appeared to me to have been the natural outflow of the epoch-making labours of Dr. John Snow on the relation of epidemic cholera to infected

water, and the subsequent demonstration by others of a similar origin in the case of outbreaks of enteric fever.

Of necessity all that could be at first established as to the spread of specific disease by milk, by the comparatively rough method of clinical inquiry, was the bare fact of the milk being accidentally infected and capable, when so infected, of conveying the specific contagium into the human system. But since the rise and cultivation of the science of bacteriology, new and more delicate methods of research have become available. We have now been taught by the experts in this branch of physiological and pathological microscopy, and by the complementary work of organic chemists, a great deal that we wanted to know about the nature of that which we were in the habit of calling "*materies morbi*," and, where this has been determined, a good deal also about the connexion that sometimes exists between an animal furnishing food and the disease which that food may produce in man. And this new method of research has further become available for the elucidation of the phenomena of what has been called "food-poisoning." I propose in this communication to lay before the Section the results of a short series of experiences which, during the last 10 or 12 years, have fallen to the lot of the Medical Department of the Local Government Board of this country, experiences which very largely derive such value as they possess from the devoted labours of Dr. Klein, who has always been obligingly ready to favour the Board and the Department with his invaluable assistance.

Fourteen instances of such poisonings out of a larger number that have from time to time come under the notice of the Medical Department of the Local Government Board are available for my present purpose, and will serve to illustrate the points to which I desire to draw the attention of the Section. Some of them have been fully reported in the published reports of the Medical Officer; others have been published elsewhere. They are as follows:—

1. In 1880.—The Welbeck epidemic, due to the eating of cold boiled ham. (Report of Medical Officer of the Local Government Board for 1880.) Investigated by Dr. Ballard and Dr. Klein.
2. In 1881.—The Nottingham series of cases due to the eating of baked pork. (Report of Medical Officer for 1880.) Investigated by Dr. Ballard and Dr. Klein.
3. In 1881.—A case of sausage poisoning near Chester. Investigated by Dr. Ballard and Dr. Klein.
4. In 1882.—An instance of poisoning from tinned pigs' tongues at Oldham. Communicated to the Department by the Medical Officer of Health, Dr. Sutton.
5. In 1882.—An instance of a family poisoned by roast beef at Bishop Stortford. Communicated to the Board by the Medical Officer of Health, Mr. Turner.
6. In 1882.—An instance of poisoning of several families at Whitchurch by brawn. Communicated to the Board by the Medical Officer of Health, Dr. Thursfield.

7. In 1878.—An instance of family poisoning by roast pork again at Whitchurch. Communicated to the Board by the Medical Officer of Health, Dr. Thursfield.
8. In 1884.—An instance of family poisoning by tinned salmon at Wolverhampton. Investigated by Dr. Ballard and Dr. Klein.
9. In 1886.—Poisoning by articles consumed at a wedding breakfast at Carlisle. Investigated and communicated to the Department by Dr. H. Barnes, of Carlisle.
10. In 1886.—Instance of family poisoning by veal pie at Iron Bridge. Communicated to the Board by the Medical Officer of Health, Dr. Thursfield.
11. In 1887.—Poisoning by pork pie or brawn at Retford. (Report of Medical Officer for 1887.) Investigated by Mr. Spear and Dr. Klein.
12. In 1889.—The Carlisle (B) case. Poisoning by pork pies and boiled salted pork. (Report of Medical Officer for 1889.) Bacteriological investigation by Dr. Klein.
13. In 1891.—Poisoning by meat pie at Portsmouth. (Report of the Medical Officer for 1890–91.)
14. In 1888.—The epidemic of pleuro-pneumonic fever at Middlesbrough. (Report of Medical Officer for 1888.) Investigated by Dr. Ballard and Dr. Klein.

It may suffice here to give a brief outline of these instances. In all but the last instance, the prominent phenomena were those of gastro-enteric disturbance; in the last instance the prominent local phenomena were those of pleuro-pneumonia. The essential character of the epidemic was a specific fever which spread through the locality from person to person and from family to family, partly through the atmosphere and sewer and drain emanations, but partly (as I think I have demonstrated) through the use of infected food.

There is a strong family likeness in the phenomena exhibited by the cases of disease observed in the first 13 instances, in both their clinical and pathological features. If the diseases induced in all were not identical, they were so nearly identical that at the present moment it is not possible to distinguish them. The phenomena exhibited were those of more or less severe gastro-enteric disturbance and those of more or less pronounced disturbance of the nervous system. The first symptoms of serious illness usually set in without preliminary warning at a varying period after the eating of the poisonous food. They were rigors or a sense of faintness with muscular weakness and prostration sometimes very severe, giddiness, abdominal pain, vomiting and diarrhœa commonly of a very offensive character; these symptoms were soon followed by fever, headache, often intense, and great thirst; and as the malady progressed other nervous disturbances were observed, such as cramps, muscular twitchings, various disturbances of vision, dilatation of the pupil, drowsiness, and even sometimes coma. Convalescence was apt to be tedious, and was sometimes accompanied by desquamation of the cuticle. The phenomena observed in the principal organs of the body after death were inflammatory, hæmorrhagic or

destructive changes in the stomach and intestines, pulmonary engorgement or a hæmorrhagic condition of the lung tissues, and inflammatory or destructive changes in the liver and kidneys. These are phenomena not of mere local irritation but of a *general* disease having alliances with our ordinary specific fevers.

1. The Welbeck Epidemic occurred in 1880 among a crowd of some 2,000 persons who attended a sale on the estate of the late Duke of Portland. A large number of persons who partook of the refreshments provided were subsequently taken ill, and four persons died of their illnesses. I succeeded in tracing the mischief to the eating by those attacked of certain hams made in this country from salted pork imported from America: they were cooked in the dirty and unwholesome kitchen of an hotel, where they were stored for more than two days before they were eaten. The time which elapsed before attack varied: in five out of 51 cases in which this point could be determined it was 12 hours or less, in 34 cases between 12 and 36 hours, in eight cases between 36 and 48 hours, and in only four cases longer than this. In the residuary cooked ham, and in raw ham made from the same consignment of pork and at the same establishment, Dr. Klein discovered a species of bacillus which he has fully described in the appendix to my report upon the outbreak. Feeding and inoculation of dogs, cats, and rodents with both of these materials and with the culture material gave positive results. With few exceptions the animals were taken ill, and from such of these as died or were killed during their illness the bacilli were recoverable. In the kidney of one man who died after about a week's illness there were evidences of parenchymatous inflammation, and the afferent arterioles and capillaries of the Malpighian corpuscles were found plugged with emboli formed by masses of the bacilli. There were indications in this instance that all parts of the same cooked ham were not equally poisonous, and that the poisonous quality of the material became more pronounced as day succeeded day (for five or six days) after cooking. In a portion of cooked ham exposed to sewer air for some hours, although the bacilli were discoverable, the material had ceased to be poisonous to animals fed or inoculated with it.

2. The Nottingham series of cases occurred in 1881. In this instance several distinct families partook of a certain roast leg of pork dispensed in slices with gravy at a shop in the town of Nottingham. Such members of the several families as partook of the pork were taken ill, while those who did not partake of it escaped. One of the sufferers died after about four days' illness. The period that elapsed between eating and the first definite symptoms of illness varied from 12 to about 34 hours. Bacilli similar to those found in the Welbeck instance were found by Dr. Klein in the blood, in the lungs, and in various abdominal organs of the man who died, and rodents inoculated with the blood, pericardial exudation, and lung juice became diseased and died, the bacilli being recoverable from their blood and exudations. No one was taken ill from eating cold pork (served without gravy). In this instance I had reason to suspect that it was not the pork itself which was the peccant material, but the gravy which was served with it. The

reasons for this suspicion are fully stated in my published report. The gravy was merely some melted jelly or stock prepared in a filthy cellar by boiling pork bones, pigs' feet, &c., and stored there until required for use, when a portion was taken away and warmed to about 144° Fahr. In this instance, again, all parts of the pork or jelly from which the gravy was made were not equally poisonous.

3. The Chester Case.—Shortly after this I was instructed to investigate the circumstances of the death near Chester of a man who, about half-an-hour after eating some so-called "American sausage" (a material which arrived packed in a tin and consisted partly of pork), was seized with very violent gastro-enteric symptoms and great prostration, and who ultimately died after a few days apparently with severe pneumonia. No post-mortem examination was obtainable, as I could not obtain the concurrence of the coroner, but the remains of the sausage partaken of, together with other sausages of the same consignment, were sent to Dr. Klein for experiment. Some portions of the actual sausage eaten killed animals fed with it, while other portions were innocuous. Post-mortem examination of the animals that died showed hæmorrhage into the stomach, congestion of the lungs, and hyperæmia of the medullary portion of the kidneys; the most important fact, however, about these organs being that most of the urinary tubules contained casts, while many of the Malpighian corpuscles with their surrounding tissues were in a state of disintegration without, however, any inflammatory cells being present, indicating the disintegration to be the direct result of some destructive agency circulating in the vessels.

4. The Oldham Case.—In this instance American tinned pigs' tongues were concerned. This material was partaken of at dinner by members of two families. The portions thus eaten were cut from the top of a freshly opened 7-lb. tin, no more of the contents of the tin having been used except for purposes of analysis and experiment. Only those members of the families who ate of the food were taken ill. They mostly felt nauseated speedily, but the more marked symptoms occurred after the lapse of about four hours, except in the case of the father of one of the families, who only tasted the food at the end of a knife—his illness was delayed for eight hours, but lasted longer than that of his children who ate more. The most serious case, which became comatose, was one in which diarrhœa was absent; in this case signs of recovery did not appear until the bowels had been acted upon by a purgative. What was left in the tin after the chemist to whom the material was referred had done with it was sent to Dr. Klein, who fed and inoculated a variety of animals with it without injury. Either the poison had disappeared from the material by keeping for a few days, or it had been confined to that end of the tin which was first cut from.

5. The Bishop Stortford Case.—This is a case in which members of three families who ate of a particular piece of ribs of beef (the rest of the carcase proving harmless) when it had become cold (not when hot, freshly cooked) were attacked. The meat was cooked on a Saturday, and its poisonous quality was more marked on the Monday

than on the Sunday. There was a considerable interval (incubation period) between eating and illness; in one family in which the meat was eaten cold on the Monday at breakfast the illness did not commence until the next day. No bacterial investigation was made.

6. The Whitchurch "Brawn" Case.—Brawn is a gelatinised dish made principally of pig's head, and eaten cold. In this instance brawn, made and purchased at two different shops between which there was no connexion except that they were situated in the same street, poisoned nearly simultaneously members of 10 different families residing in different parts of the town. The brawn does not appear to have been kept under conditions specially favourable to decomposition or contamination. At both shops the brawn was made on September 7th, taken into the shops for sale on the 8th, when some of it was eaten by purchasers; but the large majority of the families that are stated to have been taken ill after eating it did not eat of it until the 9th. Only one family that ate of it on the 8th is mentioned as having suffered. The attacks commenced suddenly from one to four hours after eating. In some prepared microscopical specimens of the material forwarded to him, Dr. Klein found bacilli looking like those he found in the Welbeck hams, but he could not say that they were identical with them.

7. The Whitchurch Roast Pork Case.—This instance of food poisoning occurred also in the town of Whitchurch, but in 1878. The material which produced the illness was a roast leg of pork (the remainder of the carcase having done no mischief). The pork was provided for a Sunday family dinner, at which it was eaten freshly cooked and hot; it was eaten cold at supper and cold again at dinner on Monday; it was eaten also cold by a man and his wife residing three miles off, both of whom died from its effects after about 30 hours illness. The others who were attacked recovered. There appears to be evidence that the pork eaten, whether hot or cold, on the Sunday did no mischief. It was not until the Monday that it made people who ate it ill. And there is this further interesting and instructive fact to be noted, viz., that those who ate the pork at dinner only on the Monday were not attacked until after an interval of seven to 19 or more hours, while the two persons who ate it in the evening and died were attacked much more quickly, namely, about four hours after eating.

8. The Wolverhampton Tinned Salmon Case.—In May 1884 three grown-up persons at Wolverhampton ate at supper a portion of a tin of salmon, and two children merely tasted it. The tin was what is termed "a blown tin," and when opened the material was found to have been in some degree decomposed. The son who had eaten most was attacked about 10 hours after eating it, and died in three days; the mother who ate less was attacked after 12 hours, and died in five days, and the daughter, who had eaten still less, was attacked after about 14 hours, but recovered. The two children suffered merely from transient headache the next day. Portions of the stomach, liver, and kidneys were submitted for examination to Dr. Klein. In both fatal

cases the stomach showed necrosis of the superficial layer of the mucous membrane, the liver extensive fatty degeneration as in acute phosphorus poisoning, and the kidney signs of inflammation. Very little of the material remained in the tin sent to Dr. Klein, but mice fed on it speedily fell ill and died, their organs being similarly affected to those of the man and woman who died, but no micro-organism was found in them or in the blood. They had obviously been poisoned by some chemical poison.

9. The Carlisle (A) Case.—In August 1886 happened what was called the "Carlisle poisoning case." At a wedding breakfast and subsequently, 24 persons in all partook of the refreshments provided, all of which were cold: they had been prepared the previous day and stored for the night in an ill-ventilated cellar where (unknown to the fresh occupants of the house) meat and milk were apt to go bad. Only four of these persons escaped illness; both bride and bridegroom were attacked, and the former died in Edinburgh. No post-mortem examination was made. Dr. Barnes's suspicion was chiefly directed to the "American ham" that formed part of the refreshments provided (mainly perhaps because he was familiar with the history of the Welbeck epidemic), but there is reason to believe that this was not the only peccant article. The gelatinous articles provided—jellies and open game pie for instance—were not improbably also in fault. Three of the 20 made ill certainly ate of the food on only one occasion, viz. the bride and the bridegroom and one other person; the bridegroom was attacked after an interval of eight or nine hours, and the bride and the third person after an interval of 17 or 19 hours. The probable interval in other persons who ate of the food at more than one meal was from 6 to 43 hours. Chemical investigation indicated the presence of a toxic chemical substance in the ham (alone submitted to investigation). No bacilli were found in the ham, but Dr. Klein and Dr. Chiene of Edinburgh obtained from it a cultivation of micrococci which (as well as the portions of the ham with which animals were fed by Dr. Walker of Newcastle) were harmless to dogs, cats, and mice.

10. The Iron Bridge Case.—In this instance 12 individuals out of 15 in a household partook at mid-day dinner of one or two veal pies, made the day before but warmed up again for the meal. Again the period which elapsed between eating the pie and commencement of illness varied. Three were attacked after six or seven hours, four after seven to 10 hours, and the remainder later in the night. The unused pie was forwarded to Dr. Klein after it had been kept for 13 days, as well as a portion of the raw veal from which it had been made. The raw veal produced no ill results on mice and a dog and a cat fed with it, nor did the pie until the feeding had been continued for three days, when two of the mice died. Post-mortem examination gave evidence of intestinal inflammation and congestion of the kidneys, but no organism was detected in the blood or viscera. This pie when opened was found to be mouldy, and there was a whitish scum which was an almost pure cultivation of a species of motile bacterium resembling the bacterium

termo, and between the pieces of meat and in the jelly of the pie were two kinds of motile bacilli. All these organisms were cultivated. No ill effects followed feeding or inoculation with the cultivations of any of these organisms except one, the special kind of bacterium termo just mentioned. This bacterium and its culture characteristics are fully described by Dr. Klein in the Report of the Medical Officer of the Board for 1890 (p. 194). One curious thing about it is that it does not grow well in any medium at a temperature above 30° or 32° C.; at 36° to blood-heat no growth takes place. A very interesting fact about all its cultures is that after some days' growth the cultures possess a most exquisite and delicate aromatic odour, no trace of putridity being perceptible. Experiments made with the pure cultivation of this bacterium resulted as follows:—As might be expected from the fact that it does not grow at blood-heat, subcutaneous inoculations into mice produced no result, but when mice were fed with the contents of a culture tube they fell ill and died, the customary lesions in the stomach and intestines, lungs, liver and kidneys being found after death, but no bacterium termo was to be found in any of the viscera. The obvious inference from this is that the cultures of this bacterium contained a substance which, when introduced into the stomach, produced illness and death, in the latter event severe gastro-enteritis being a conspicuous feature. Since the organism in itself is harmless when inoculated, not being capable of growth and multiplication at the temperature of the animal body, it follows that the substance which effected the poisoning was non-organised and produced by the above bacterium termo.

11. The Retford Case.—November 1887, 80 persons in 22 different families were made ill, and one of them died from eating pork pie or "brawn" bought on one or other of two days at some co-operative stores there. The pork was cut up on the 9th, the pies and brawn were cooked on the 10th, and they were eaten on the 11th, 12th, 13th, 14th, and later. It is to be noted that, with the exception of one family, none who ate of this food-stuff on the 11th (the first day after cooking) were taken ill; and further that none who ate of it after the 14th (the fourth day after cooking) were taken ill. Here again the interval between eating and illness was various. Out of 59 cases in which it was determined, 4 individuals were attacked after an interval of less than 8 hours, 6 after an interval of between 8 and 12 hours, 19 between 12 and 24 hours, and 30 between 34 and 36 hours after eating. Unfortunately the report does not state whether those who were attacked early were of the families who partook of the food sooner or later after it was cooked. One of the pork pies forwarded to Dr. Klein on December 1st (three weeks after cooking) was found to contain a brown viscid scum "made "up almost entirely of minute thick rods, some of which were motile or "of the kind now spoken of as short bacilli," the streak cultivation of which on nutritive gelatine begins to assume after 24 hours a blue colour, which Dr. Klein says is extremely characteristic. It was not the same bacillus as was found in the Iron Bridge case (No. 10), partly differing from it in this, that it readily grows at the temperature of the body. Feeding with the pie made six mice ill and killed one of them, as also

did feeding with the cultivation of the bacillus, post-mortem examination revealing gastro-enteritis with hæmorrhage and congestion of the spleen, liver, lungs and kidneys: similar results followed inoculation with a three-days' old cultivation of the bacillus on gelatine. Cultivation of the heart's blood in tubes of nutritive gelatine yielded no growth of any kind; but in one instance where this failed, the bacillus grew copiously on using some of the exudation into the pleura, and on the inoculation of these cultures similar effects were produced in other mice. In connexion with this culture a very striking difference was met with, viz., that the material of cultures 10 days to a fortnight old was absolutely without effect on mice fed or inoculated with considerable quantities of it. This was proved not to be due to the death of the bacillus, but it would appear that the chemical substance poisonous to mice is produced by the bacillus only at an early period and for a very limited time in these subcultures. We may note in connexion with this fact the other observation made by Mr. Spear who reported upon the case, that persons who ate of the foodstuff after the fourth day from cooking were not made ill by it.

12. The Carlisle (B) Case.—During the first week or 10 days of November 1889, a number of persons of different families, some residing within and some outside the city of Carlisle, were made ill by eating pork pies or boiled salted pork purchased at one or other of five establishments in different parts of Carlisle. Two of these establishments belonged to a butcher who made pork pies and sold also boiled salted pork; the other three establishments belonged to a cocoa-house company, who purchased pork of this butcher, and had pork pies made of it at their bakery from which their three shops were supplied. The particular pork which produced mischief appears to have been made into pies on Friday, November 1, having been killed two or three days previously. The people, numbering 25 or more, who were taken ill, appear to have eaten of one or other of these materials at dates from November 1 to November 11; and very generally there was an interval of not less than 24 hours between the eating of the food and the commencement of serious illness. This is as much information of the circumstances of the outbreak as can be gathered with precision of detail from the account of it in the Board's possession. The medical officer of health transmitted to Dr. Klein on November 25 samples of salted pork from all the pigs the butcher had slaughtered from October 28 to November 4, and a sample of gravy-stock used by him in making pies. None of the pork pies of the butcher which appeared to have been poisonous, were obtainable. It must be added that it could not be affirmed that the gravy-stock, which was sent to Dr. Klein, was the same as that which had been used for the particular pork pies which had seemed to produce illness. Mice fed with the pork, or with the gravy-stock, on the second or third day from eating, had diarrhœa, with fluid sanguineous evacuations, and died, and presented identical *post-mortem* appearances. The whole of the small *intestine* was relaxed and congested, and its cavity was filled with sanguineous mucus. Both *lungs* were deeply congested, and in the mice that

survived longest there was hepatisation chiefly in the upper lobes. The *liver* was congested; in one animal only (a mouse dying on the sixth day after feeding with the pork) were there present numerous whitish grey specks in that organ. The *spleen* was congested in all; in a few cases only was it in addition very slightly enlarged.

Gelatine cultures inoculated with juice from the diseased lung, afforded in the case of every one of these mice an organism which proved to be a minute motile bacillus, which is fully described in Dr. Klein's published report of his observations (p. 224). The following is Dr. Klein's summary of the results of his experiments:—

"1. The stock-gravy and the pork were alike capable of producing illness and death in mice fed with these materials.

2. The illness and death thus produced could hardly have been due to a chemical poison, for the induced disease did not set in until the second day after experimental feeding; there was, in fact, a definite incubation period of not less than 24 hours in all cases.

3. The symptoms noticed during illness, and the appearances observed after death, indicated severe intestinal disturbance and congestion of the lungs.

4. In the congested lung were present numerous bacilli of a single species that had definite cultural characteristics.

5. The bacillus thus obtained when introduced, whether by feeding or by inoculation, into other mice, produced in them illness and death,—with the same symptoms and pathological appearances as had been observed in mice fed directly with the pork or with the gravy-stock under suspicion. And further, from the bodies of the mice dead after feeding or inoculation with cultures of the bacillus, that organism was recoverable in abundance by appropriate methods.

To sum up. We have then to do here with a true infectious disease producible in the first instance by feeding animals with given samples of gravy-stock and of pork, and capable of being propagated by a particular microbe isolable in artificial culture from the bodies of the rodents fed with the samples. The microbe here described is not the same as that described by me 10 years ago in connexion with the Welbeck and Nottingham meat poisoning; nor is it identical with either of those to which veal and pork pie poisoning has been referred by me in recent reports to the Medical Officer. It is an altogether different species, as will be clear from my account of its morphological and cultural characters."

Dr. Klein further adds that he was able to isolate the microbes from the gravy-stock, but was less fortunate in the case of the pork which had become mouldy by the time it had reached him.

13. The Portsmouth Case.—One afternoon in February, 13 persons, nine boys and four grown-up persons, partook of cold meat (beef) pie, made and cooked the day before. One of the boys ate none of the meat but only the paste and gravy, and one grown-up person

scarcely touched what was given him. The last-mentioned person escaped illness, but all the others were taken ill in the night or early morning, *i.e.*, 14 to 17 hours after the meal, with abdominal pains, diarrhœa, headache, and malaise: all recovered. The same paste used for treacle pie and tartlets eaten by other persons had produced no mischief, so that the peccant material would appear to have been the meat or the gravy; the chief suspicion falling upon the gravy, as one boy who ate only paste and gravy became as ill as the rest. Portions of the cut pie and of one meat pie that had not been partaken of with a good deal of adherent jelly were submitted to Dr. Klein for experiment. The pies when first received proved poisonous to mice, but five days later, when the material had become offensive from putrefaction, it was poisonous no longer. Microscopical examinations of the jelly showed two species of bacilli, one, *a* non mobile, broth cultivations of which had a rather pleasant aromatic odour, and the other, *b*, mobile and rapidly liquefying gelatine and producing an offensive odour. By direct experiment, feeding mice on broth culture of these bacilli, the culture of species *b* was found to be harmless; but that of species *a* was found poisonous, and certain of the mice thus fed died. Inoculations of the broth culture of species *a*, however, had no effect. In the dead animals the lungs were found dark red and almost hepatised, the liver, spleen, and kidneys dark, spleen not enlarged: the small intestine relaxed and full of mucus. From only one of the mice that died could the bacillus be recovered. The inference drawn by Dr. Klein is that the bacillus itself was not pathogenic on inoculation but that what produced illness and death was a chemical poison formed in its culture. He adds the remark that this organism *a* is morphologically and culturally different from the species found in the Retford, Shrewsbury (Iron Bridge), and Carlisle (B) materials respectively.

14. The Middlesbrough Pneumonia Epidemic.—This epidemic which prevailed during the early part of 1888, and resulted in 490 deaths during the year in a population of about 98,000 persons largely composed of iron workers, was intrusted to me for investigation. The prevalent disease was proved incontestably to be of infectious character, communicable from person to person by proximity, through the medium of sewer-air, and in other ways in which other infectious fevers are known to be spread. But all this left unexplained a curious geographical limitation of the epidemic which puzzled me greatly, until calling to mind some old experiences I began to study the food and mode of life of the classes principally attacked. I then obtained the clue that I wanted, in the double fact that during the greater part of every week nearly the only animal food these people got was what is termed "American bacon," made by soaking in water and then only slightly drying salted pork imported from America; and that the limitation of the epidemic corresponded closely with a similar geographical limitation of the wholesale trade of a manufacturer of this bacon in the town of Middlesbrough, who in the affected districts had by far the largest portion of this trade in his hands. This was the thread which, by following it along, led me to the discovery I am about

to mention. I sent to Dr. Klein for examination and experiment fresh portions of viscera (as well as hardened portions) of persons dead with the malady, and also specimens of the suspected bacon of which sick persons had recently eaten, and similar specimens purchased at different retail shops in the district. The result of Dr. Klein's work upon these materials was briefly this :—That the disease I was dealing with was, as I suspected, no new local malady, but a specific general disease or fever marked, as other general specific diseases are, by destructive morbid changes in all the principal viscera, the special characteristic of which in this case was a pleuro-pneumonia. In the lung juice and in the lung tissue Dr. Klein discovered a hitherto undescribed short bacillus which he has called "bacillus pneumoniæ," differing altogether from the bacillus of Friedländer and from the "diplococcus pneumoniæ" of Fränkel and Wiechselbaum, neither of which was present. Of 20 samples of bacon forwarded from the infected districts, 14 were distinctly poisonous to rodents fed with it, in two instances there was some doubt, and only four proved not be poisonous. In the dead animals lung lesions and lesions of other viscera similar to those observed in persons who died of the disease in the infected districts were found. Similar results followed inoculation of the human lung juice and of pure cultivation of the bacillus pneumoniæ. In all instances the bacillus was recoverable. An instructive incident in Dr. Klein's part of the investigation was this, viz., that during its progress an epidemic of pneumonia occurred among the animals, mice, guinea pigs and monkeys kept in the building where his experiments were carried on, the bacillus pneumoniæ being found after death in the lung juice and sometimes in the heart's blood also. Another fact must be mentioned, namely, that on re-examination after the lapse of three months, of samples of the bacon that had previously produced illness and death, they were found to have lost their powers of infecting animals, and no growth of the bacillus was obtainable. The details of the evidence on which I base this connexion between the use of the suspected kind of bacon and the spread of the epidemic disease will be found in my detailed report on the epidemic in the 18th Annual Report of the Medical Officer of the Local Government Board.

This is the first instance, that I am aware of, in which food has been even suspected to have been concerned as an agent in the production or spread of pneumonia. It was found in the course of the inquiry, that mice fed upon the sputa of the sick fell ill with the disease and died, the bacillus being recoverable from their bodies. Hence for the recognition of this form of pneumonia, where precautions against its spread in a family or neighbourhood ought to be taken, I have in this country where legislative restrictions deprive ordinary medical practitioners of the advantages they might otherwise confer upon the public by an occasional test-inoculation, been advising medical men to make use of the feeding of mice with pneumonia sputa as a means of diagnosis.

The following inferences appear to flow from the instances of food-poisoning thus briefly outlined, and from investigations pursued under

the auspices of the Local Government Board by Dr. Macfadyen and others, and recorded in the reports of the Medical Officer.

1. I may quote the words of Dr. Buchanan the Medical Officer of the Board. He says (19th Report, p. xiii.):—"The phenomena which "we speak of as 'food-poisoning' productive now of one and now of "another sort of definite malady among consumers of certain foods are "claiming, on ever-growing evidence, to be regarded as true infective "diseases, as much so as scarlatina or tuberculosis. That they have "not been generally admitted into this rank arises, first, from the "circumstance that some of them have seemed to be wanting in an "incubation period; and secondly, because they are rarely recognised "as transmissible from person to person. But from our researches of "this very year (1888) we derive some new considerations that "materially affect the importance of incubation, as determining the "infective nature of a poison; while we need go no further than "the experiences of Middlesbrough in 1888 to find suggestion of "disease-bacteria operating alternately through the atmosphere and "through infection of food material by them."

2. In infected food capable of producing disease on being eaten we find one or both of two things—a living microscopic organism and an organic chemical poison of greater or less virulence.

3. Of these two things, that which is immediately operative in the production of the morbid phenomena is the chemical poison which is apparently of a basic nature and a product of the processes of bacterial life.

4. Specifically different bacteria capable of producing this chemical poison may through its agency give rise in the human system and in animals to clinical phenomena and pathological changes in the organs which are so similar that at present they cannot be distinguished.

5. Given the bacterium and favourable environment, the bacterium may grow, multiply, and produce its own special chemical poison from the material which affords it nourishment either outside the body or within it. One important element of environment is temperature. If a bacterium will not grow at the temperature of the body, it of course will not produce its operative chemical poison when introduced into the system.

6. Moreover, both the bacterium that produces the chemical poison in an infected food and the chemical poison itself may apparently be evanescent; perhaps in the former case the bacterium being killed by its own products, and perhaps in the latter case, because the chemical poison undergoes destructive changes. Hence an infected food, poisonous when eaten at one time, may fail to be poisonous when eaten at a later period; or poisonous when inoculated at one time, may cease to be so later on; or one portion of an infected mass of food may be poisonous, and another part not be so.

7. It is to be observed that in many cases of food poisoning an incubation period has been distinctly traced, in others it has been less obvious; in some there was practically none, only from half-an-hour

to a few hours elapsing between taking the food and the initial symptoms of the malady. This presence and absence of incubation, may be taken as clinical evidence of the symptoms being due to the operation within the body of the bacterium itself, or of their being due (at first at all events) to the operation of the chemical poison already prepared in the food. Where merely the bacterium is introduced, time is required as in other specific infections for the growth and multiplication of the micro-organism in the body, with the formation there of its poisonous chemical product. When the chemical poison already prepared outside the body is introduced, it operates more speedily, the rapidity of the operation being proportional to its quantity and quality, and to the individual peculiarities of the recipient. In the several series under consideration, the instances of long incubation (where it has been possible to ascertain the fact) occurred in cases where there was no doubt of bacteria having been introduced into the system, as in the Welbeck and Nottingham ham and pork cases, and the Retford pork-pie case. I had reason to think that in the case of the Middlesbrough bacon the incubation period was 18 to 36 hours, or perhaps a little longer. On the other hand, where it was certain that no bacterium was introduced in the food or one which would not grow at the temperature of the body—as in the Chester sausage case, the Wolverhampton tinned salmon case, and the Iron Bridge veal-pie case—or was non-pathogenic on inoculation as in the Portsmouth instance, the illness commenced at periods varying from half-an-hour to 10, 12, 14, or 17 hours only after eating. But in some of these food-poisoning series of cases there were individuals who suffered quickly and others who suffered after a longer interval. It was so in the Welbeck series of cases, where the interval varied from less than 12 hours to more than 48 hours, in the Nottingham pork case, where it varied from about 12 to 34 hours, in the Whitchurch pork case (No. 7), from seven to over 19 hours, in the Carlisle (A.) case from six to 43 hours, and in the Retford case from under four to about 36 hours after eating. The interpretation of these varieties appears to be that, in the food eaten, the bacterium capable of developing in the body and the chemical poison already formed in the food were not equally distributed through the mass of the material, one individual case getting more of the one and another more of the other in the portion given him. Where, as in the Whitchurch pork case (No. 7), persons who fed upon the food latest had the shortest interval and the most severe attack, the interpretation may be that the individuals who fed at an earlier period got the bacillus in an active stage, when little of the chemical poison had been produced in the food, and those who fed later on got a larger dose of the chemical poison, for the more abundant production of which by the bacterium time had been afforded by the postponement of the eating.

8. As regards the kinds of animal food which in the adduced instances produced mischievous or fatal results:—Of the 14 instances food was or consisted largely of—

Pig-meat of one kind or another in 9 instances.
Veal - - - 1 instance.

| | |
|---|---|
| Beef - - - - | 1 instance. |
| Butcher's meat (kind not stated) | 2 instances. |
| Tinned salmon - - | 1 instance. |

I am disposed to think that this is no unfair representation of the relative frequency with which swine's flesh thus gives rise to specific diseases of the kinds referred to, as compared with animal food from other sources. Certainly, apart from these instances, this corresponds with my general experience, and I suspect with the experience of the profession at large. What explanation can be offered of this? There is no reason whatever to believe (quite the contrary) that the food in any of the instances above recorded contained the specific micro-organism before it was prepared for food. The American pork which, converted into mild bacon at Middlesbrough, was a means of conveying to persons eating it a specific pneumonic fever was obtained from American firms at Chicago and elsewhere of the highest repute, and consignments of the same brand at about the same time to other similar bacon-makers at Stockton, only a mile or two distant, did not spread any epidemic, so far as I could learn, among its consumers; nor did other hams or bacon than those which produced disease at Welbeck, although made from the same consignment of American pork, produce similar disease to the Welbeck epidemic. And it is to be noticed that in the Nottingham pork case and the Bishop's Stortford beef case (No. 5) other portions of the carcases were eaten harmlessly. And this is an observation I have made in other instances of food-poisoning within my experience.

The truth of the matter seems to be that the accession of the specific bacillus to the food is what we commonly call "accidental," which of course only means in this case that it comes from somewhere beyond our present knowledge. We do not know its normal habitat, but assuredly it now and then becomes air-borne, infects food of a kind that affords it appropriate nutriment, multiplies and produces its appropriate chemical poison there, and produces its appropriate specific disease in persons who consume the infected food. I have elsewhere suggested a similar hypothetical explanation of epidemic summer diarrhœa, another unquestionable specific disease. But if we do not know where the normal home of the bacillus is, nor how it escapes to do mischief, we do know something about the circumstances under which it gains access to food, which may help to elucidate even this point. It is to be noted that in every instance except one (No. 2, the Nottingham pork case), it was food that had been prepared a day or longer previously to its consumption, stored in one way or another, and then eaten cold, which produced disease. And it is the conditions under which the preparation of the food took place, and under which it was subsequently stored, that, in certain of the instances narrated, served to furnish a clue to the knowledge we want. The observations I made indicate an infectiveness of the place where these things were done, in association with uncleanliness of air, of soil, or of surface. I must be content to refer to my reports on the Welbeck and Nottingham cases, and to my summary of the Carlisle (A) case, as evidence of this, and notably to my report on the Middlesbrough outbreak, where the circumstances of the processes of

bacon making at Middlesbrough and Stockton are contrasted. Still, all this does not explain the special liability of pig-meat to produce these specific maladies. How is this to be accounted for? I have been disposed to gather a suspicion of what the explanation is from what I have said above about the Nottingham pork case, viz., that it was perhaps the gelatinous gravy, and not the flesh of the pork, that was really the peccant material. I could not hear of anyone having been made ill by eating the pork served to them after it had become cold, which, of course, would have been without gravy. And the explanation I have to suggest is this, that of all adult flesh-meats ordinarily eaten, pork under the process of cooking furnishes the largest proportion of gelatine. Young meats, such as veal (used in the Iron Bridge case), are also largely productive of gelatine; and gelatine is a favourite nutriment of morbific bacilli. We may note here how many of the articles of food most generally partaken of at the Carlisle wedding breakfast were gelatinous or gelatinized. These were the cold American ham, which moreover was glazed on the surface with gelatine, the game pie, the cold boiled salmon, and among the sweets the jellies and the trifles Of two or more of these everyone that was taken ill had partaken. The Portsmouth case illustrates the same point, since a boy who ate nothing but gelatinous gravy (with pie-crust that was beyond suspicion) was made as ill as others who had eaten the meat of the pie.

9. And yet another word before I conclude on the practical application of all this. What does it all indicate as an efficient precaution against "food-poisoning"? Of course no one is likely to be hindered by any fear of infection from eating cold ham or gelatinised food of any kind if he likes them. But if we do eat ham or bacon, cold or warm, it is a proper precaution to avoid them if not duly cooked throughout. The people who chiefly got pneumonia in Middlesbrough were a class who habitually only warmed the bacon they ate by slightly toasting it before a fire; they did not heat it sufficiently to kill any micro-organism; and hotel hams (indeed hams cooked at home too) are rarely thoroughly cooked. But the grand precaution of all is the very commonplace one signified by the word *cleanliness*. Every factory where pork is converted into brawn or hams ought to be so arranged that light and a draught of air can penetrate freely everywhere; there should be no corners where refuse matter can lodge and become a centre for the cultivation of morbid micro-organisms in filth; the rise of ground air should be obviated by cement under the pavement or flooring; and the place should be kept scrupulously clean and free from incursion of sewer air or putrid emanations of any kind. Kitchens, and above all pantries and places where food is stored in hotels, public refreshment rooms, or pastrycooks' premises, and in private houses, should be similarly cared for. It should be held to be part of the business of conservators of public health to see that these rules are observed, as well as the business of every master or mistress of a family.

## The Infection of Meat and Milk.

BY

Dr. VICTOR C. VAUGHAN, Ann Arbor, Michigan, U.S.A.

The infection of meat and milk may be discussed under the following heads :—

(I.) The infection may be due to a diseased condition of the animal from which these foods are obtained.

(II.) The infection may be due to the inoculation of these foods with specific pathogenic micro-organisms outside the body of the animal from which they are derived.

(III.) Meat and milk, especially the latter, are often infected with saprophytic, toxicogenic bacteria.

(I.) The transmission of disease from the lower animals to man by the employment of the flesh or milk of the former as a food by the latter has been repeatedly shown to occur. The cases so admirably studied by Gärtner, of Jena, illustrate the truth of this statement. A cow while suffering from a mucous diarrhœa was killed. Post-mortem examination revealed certain highly inflamed areas of the intestine. There was no enlargement of the liver or spleen, and the flesh being normal in appearance and odour was pronounced by the veterinarian as suitable for food. Fifty-seven persons in 25 families were made ill by eating of this meat, 12 having eaten the raw meat, one that which was partially cooked, and the remainder that which was supposed to have been thoroughly cooked. A vigorous young man ate 800 grammes of the raw meat at 8 p.m.; two hours later he began to vomit and purge, and 35 hours after eating the meat he died. Post-mortem examination revealed an inflammatory condition of the small intestine and swelling of Peyers' patches. All of those who ate of the raw meat were sick; while of those who ate only that which had been cooked, 36 were not affected. Among those who ate of the raw meat, the severity of illness was in direct proportion to the amount eaten. Thus, while 800 grammes caused death within 35 hours, as has been stated, one-sixth of that quantity produced an illness of 14 days, terminating in recovery. In many there was a marked elevation of temperature, the fever running as high as 40° C. In the severe cases, some of which were ill for four weeks, there was desquamation of the epidermis of the entire body. In the meat, and in the tissue of the dead man, Gärtner found a short bacillus, which he has designated by the name, *Bacillus enteritidis*. Susceptible animals inoculated with pure cultures of this germ died. The chemical poison was not isolated, but its existence was demonstrated by the fact that sterilized cultures injected into animals caused sickness and death. There can be no doubt that in these cases the flesh of the infected animal produced the peculiar and characteristic illness of those who ate of it.

The outbreak of meat-poisoning investigated by Gaffky and Paak most probably belongs to this class also, though it was not positively

shown that the horse from which the meat was obtained was sick, and it is possible that the infection might have occurred in the shop of the butcher. No person who ate of this meat escaped altogether, and one death occurred. The sausage prepared from the liver and muscles, unlike the meat examined by Gärtner, was not normal in appearance; it was greenish-yellow, sour, and nauseating. According to the man who sold the meat, the sausage was prepared from the flesh of three different horses, at least one of which was said to have been sick. In the majority of the persons who ate of this meat the symptoms manifested themselves within six hours, and in one instance within half an hour. The most prominent symptoms were headache, loss of appetite, pain in the bowels, vomiting, and purging. In the fatal case, however, there was no vomiting. From the sausage, Gaffky and Paak isolated a short bacillus, which, when given by the mouth, subcutaneously, or intravenously, produced the above-mentioned symptoms, with a fatal termination in most instances in rabbits, guinea-pigs, mice, and apes. The chemical poison was not isolated.

In the well-known Welbeck cases, Ballard has given us a valuable and detailed description of meat-poisoning,* and all of us are acquainted with the bacterial discoveries made by Klein in the ham eaten by these people.

That chemical poisons may be transmitted from the lower animals to man in the food is shown by the history of poisoning with mussels and with fish. As early as 1827, Combe described in detail the symptoms induced by the eating of poisonous mussels, and a valuable contribution to the same subject has recently been made by Schmidtmann, who has found that non-poisonous mussels placed in the water of Wilhelmshaven soon become poisonous, and that the poisonous mussels from the harbour soon lose their harmful properties when placed in the open sea. Linder has found in the water of this bay and in the mussels living in it a great variety of protozoa, amœbæ, bacteria, and other low forms of life, which are not found in the water of the open sea, nor in the non-poisonous mussel. He has also found that if the water of the bay be filtered, non-poisonous mussels placed in it do not become poisonous. He therefore concludes that poisonous mussels are those which are suffering from disease due to residence in filthy water. The discovery of mytilotoxine by Brieger has made us acquainted with the nature of one of the chemical poisons developed in the mussel and capable of seriously affecting man.

While many species of fish are popularly regarded as poisonous, but little scientific work has been done on the subject, and we are not prepared to say to what extent this popular idea is correct. Miura and Takesaki find that the ripe ovaries of *tetrodon rubripes* contain a substance which induces in rabbits acceleration of the respiratory movements, paralysis of the skeletal muscles, mydriasis, increased peristalsis of the intestines, and arrest of the heart.

---

*Vide antea*, Volume III., page 105 *et seq.*

The disease known to the Japanese as "kakke," which is said to be identical with "beri-beri," is prevalent at Tokio from May to October, and is, according to Miura, an intoxication due to the eating of fish which belong to the scombridæ. The affection is generally chronic or sub acute, seldom acute. The most characteristic symptom is paralysis of the diaphragm with consequent dyspnœa and disturbance of the action of the heart.

The question concerning the transmission of tuberculosis from cows to man through the eating of the flesh or the drinking of the milk of the former by the latter is one of great practical interest, but to what extent such transmission actually occurs I am not prepared to say.

I certainly would be willing to endorse the following propositions :—

(*a.*) The flesh of a tuberculosis cow, even when the disease is localised in the lungs, should not be eaten by man.

(*b.*) When the tuberculosis is general, there is danger of specific infection through the eating of the flesh or the drinking of the milk.

(*c.*) When there is tuberculosis of the udders, the specific infection may be transmitted through the milk.

Koch thinks that the milk contains the bacillus only when the milk-glands are affected. On the other hand, Bollinger and others find the milk infected even when the disease is confined to the lungs. This may be true; I am not prepared to deny it; but, if true, it would seem that the transmission of tuberculosis from the tubercular mother to her nursing child would be more common than we have any reason to believe that it is, unless there be some marked difference in the distribution of the bacilli in human and bovine tuberculosis. However, this point is of more scientific than practical interest. Apart from the danger of specific infection, all will agree that the flesh and milk of tuberculous animals do not furnish desirable material for the building up of the tissues of man. That lifeless proteid matter has properties of a marvellous nature has recently been demonstrated in the study of the newly discovered bacterial poisons, and Prudden has shown that dead tubercle bacilli injected into animals may lead to the development of new formations closely resembling, if not identical with, tubercular tissue.

The relation of a disease in the cow to scarlatina in children and its transmission from the cow to the child through the milk, as illustrated in the studies of the Hendon cases by Power and Klein, form another iustance of the kind of infection which we are discussing. There is one point brought out in the report of these gentlemen which deserves to be constantly borne in mind. This is that a cow which to the unprofessional observer appears to be in health, feeds well and gives milk abundantly, may still be a source of danger. This emphasises the need of having all the animals of our large dairies carefully inspected at frequent intervals by competent veterinarians. When this is generally done, and when all animals whose flesh is to be used as food undergo careful inspection before being slaughtered, we shall know much more than we now do concerning the transmission of disease from animals to man.

(II.) The infection of meat and milk outside the body of the animal with specific pathogenic bacteria is so well known to occur that I will do scarcely more than mention it. The frequency with which typhoid fever, diphtheria, and other infectious diseases are disseminated by the use of milk infected in this manner is shown in the current records of medical literature. Milk has been frequently diluted with water containing the germs of typhoid fever, and the prevalence of the disease may mark the daily rounds of the milkman. I have here a culture-tube containing a bacillus which I found simultaneously in the water from the dairy-well and in the milk from the cans. At the same time one or more cases of typhoid fever existed in every family which patronised this milkman. The bacillus resembles, but is not identical with, that of Eberth. It is highly pathogenic to white rats and guinea-pigs. I have also in another tube some of the chemical poison which this bacillus elaborates and to which its pathogenic properties are due. This poison belongs to the bacterial proteids, and from its general properties and ultimate composition I think that it should be designated as a nuclein. It was obtained in the following manner: Flasks of sterilized beef-broth were inoculated with pure cultures of the bacillus, and then kept in an incubator at a temperature of 36° C. for 14 days. At the expiration of this time, the contents of the flasks were filtered through Chamberland filters and the germ-free filtrate was precipitated with absolute alcohol. The precipitate was purified by being repeatedly dissolved in water and reprecipitated with alcohol. Finally, it was collected on a fast filter, washed first with alcohol, then with ether, and dried in vacuo over caustic potash and paraffine. The poison is soluble in water, forming a distinctly acid solution, and this injected into white rats produces the same effects which follow inoculation with the living germ. This proteid contains no sulphur, and an analysis by my colleague, Professor Freer, shows that it contains of carbon 48·46 per cent.; of hydrogen 7·69 per cent.; of nitrogen 13·44 per cent.; of phosphorous 0·69 per cent. At 55° C. it loses 8·4 per cent. of water, and at 100° C. 7·06 per cent. additional.

It is unnecessary for me to dwell upon this part of the subject, since all admit that the infection of these foods outside the body with specific pathogenic germs frequently occurs and is accountable for certain epidemics.

(III.) I wish to direct my remarks more especially to the infection of meat and milk with saprophytic, toxicogenic germs. I desire to emphasise the fact that these foods, even when derived from perfectly healthy animals, and when kept free from infection with specific pathogenic bacteria, may and often do develop most potent poisonous properties. It is not necessary that food be infected with some specific micro-organism before it can be rendered unfit for use.

It may be well to briefly discuss the nature of some of the germs with which meat and milk may become dangerously infected outside the animal body, and also the nature of the poisons which are produced by these germs. Unfortunately this class of micro-organisms has not been studied with the thoroughness and detail which it deserves. From the

studies which have been made, however, I think that we are justified in drawing the following conclusions :—

(*a.*) Many of the bacteria with which meat and milk become infected belong to the saprophytic organisms. I mean by this that these germs have not been derived, either directly or indirectly, from a diseased animal. I will illustrate this by reference to cases of poisoning from frozen custard, which I had an opportunity of investigating a few years ago. The milk supplied to a small village had never been questioned. It was in constant use by some 50 or more people, and no cases of illness had arisen which could be in any manner attributed to the milk. In the preparation for a festival some gallons of this milk were obtained and made into custard. The custard was divided into two portions, one of which was flavoured with lemon and the other with vanilla. The lemon custard was eatén without harmful effect, while a teaspoonful of that flavoured with vanilla caused nausea, vomiting, and purging. Of course it was quite natural to conclude that the vanilla was the poisonous agent, because at first it seemed that the only difference between the samples was that due to the use of the flavourings. Fortunately, however, not more than half of the vanilla in the bottle had been used, and the non-poisonous character of this flavouring was demonstrated by a young man who took two teaspoonfuls of the remainder without being in any way affected. The real difference between the portions of custard is explained by the following: the lemon custard was frozen immediately and was sent to the festival, while the vanilla custard stood for two hours before being frozen in a very filthy room, the air of which was said to have been like that of a privy vault. This room had some weeks before been used as a butcher's shop, and had never been cleansed, and the bits of decomposing meat rendered the air foul and supplied the germs with which the custard was infected.

Another illustration of this kind of infection may be found in the report by Barnes of food poisoning at a wedding breakfast at Carlisle in 1886.*

In these illustrative cases, the facts that the germs did not originate in any specific disease and that they grew in the food before it was taken into the body of the consumer demonstrate the correctness of the proposition which I have stated. But it may be asked, how is it possible for a truly saprophytic germ to induce disease and death? This may occur in either of two ways: (1) the poison formed in the meat or milk may be the sole and sufficient cause of the symptoms and death. Let me point out here the fact that the distinction between intoxication and infection is not so easy and certain as we have assumed. Especially is this true, when the agent of intoxication is a bacterial proteid. It is customary to pronounce those cases in which the symptoms occur immediately, or within less than from two to four hours, after taking the food, as due to intoxication; while those in which the first symptoms appear later are said to be due to infection. In the former the poison is supposed to be formed in the food before it is eaten; while in the

* *Vide antea*, Volume III., page 108.

latter it is supposed to result from the growth and multiplication of the germs within the body. That there is large opportunity for error in this distinction must now be evident to all who are acquainted with recent researches on the bacterial poisons. We now know that some of these poisons require a period of incubation when employed in small doses, which often extends over many days. This Brieger and Fraënkel found to be true in their study of the "toxalbumin" of diphtheria, and I have observed the same in my experiments with the products of certain saprophytic bacteria found in the stools of children suffering from summer diarrhœa, and in others obtained from drinking water. The fact, then, that the first symptoms do not appear until many hours, or even a few days after the food has been taken does not seem to be absolute proof that the bacteria continue to live and multiply within the body.

(*b.*) A germ may grow in the intestines and still be an obligate saprophyte. The food in the duodenum has no more vitality than that in the nursing-bottle of the infant. Moreover, the excretions poured into the intestines are not supposed to be possessed of vitality. A germ which will grow on a certain medium in a culture flask and produce a poison may grow on the same medium in the intestine and produce the same poison, provided it is not destroyed or modified by some secretion of the body.

(*c.*) Some of the saprophytic bacteria with which food is infected outside the body may be, and probably are under certain conditions, capable of living for a time at least in a parasitic manner. Thus, Novy has found the same germ in a poisonous cheese, and in the spleen and liver of animals which had been killed by feeding on the cheese. However, the parasitic nature of this germ, or, in other words, its capability of overcoming the resistance of the living tissue, seems to have been feeble, and instead of increasing in virulence as it was passed through successive animals it became markedly less pathogenic, and, finally, was without effect upon animals. I infer, from this and similar experiences which I have myself observed in experiments with saprophytic germs obtained from poisonous foods and from drinking water, that the toxicogenic properties of these organisms are best manifested when they are grown on dead matter.

(*d.*) The toxicogenic properties of these bacteria are also influenced largely by the conditions under which they develope. The most important of these conditions are the nature of the infected food, the temperature, the amount of oxygen supply, and the time which elapses between the infection and the consumption of the food. I have been convinced that the poisonous properties of canned meats are in some instances due to the fact that the germs which they contain grow, practically, without any air supply. The following brief report of a case of poisoning with canned salmon supports this belief:—About two months ago, Mr. K——, a very vigorous man of 34 years, ate freely of canned salmon. Others at the table with him remarked that the taste of the salmon was peculiar, and refrained from eating it. Twelve hours later Mr. K—— began to suffer from nausea, vomiting, and a griping pain in the abdomen. Eighteen hours after he had eaten of the salmon I saw the patient. He

was vomiting small quantities of mucus, coloured with bile, at frequent intervals. The bowels had not moved, and the griping pain continued. He was covered with a scarlatinous rash from head to foot. His pulse was 140, temperature 102° F., and respiration shallow and irregular. The stomach and large intestines were thoroughly washed out, and 10 grains of calomel, soon followed by 12 ounces of solution of citrate of magnesia, for the purpose of cleansing the small intestines, were administered. After these medicines had acted freely Mr. K—— began to improve. The next day the rash had disappeared, but the temperature remained above the normal for four or five days, and it was not until a week later that the gentleman was able to leave his house. I obtained the remainder of the salmon, and submitted it to various tests. In the first place the absence of inorganic poisons was demonstrated. Secondly, it was found that the subcutaneous injection of 20 drops of the fluid expressed from the salmon caused evident illness and suffering in a white rat. The only germ which could be found, either by direct microscopic examination or by the preparation of plate cultures, was a micrococcus, and this was present in the salmon in great numbers. This germ grew fairly well in beef-tea, but the injection of five cubic centimeters of the beef-tea culture of different ages failed to affect white rats, kittens, or rabbits. However, this micrococcus when grown for 20 days in a sterilized egg, after Hueppe's method of anaërobic culture, produces a most potent proteid poison. The white of the egg becomes thin, watery, markedly alkaline, and 10 drops of this suffices to kill white rats.

Évidently in the preparation of the salmon this can was not sterilized; it was sealed, and for months, possibly longer, this germ had been growing anærobically, and elaborating a chemical poison.

On the other hand, I have known several instances in which canned meats were not poisonous when first opened, but soon became so on standing exposed to the air. In these cases the meat probably becomes first infected after the opening of the can.

I will now make some general remarks concerning the nature of the chemical poisons formed in meat and milk as a result of infection with saprophytic bacteria. First, the ptomaines which are formed in foods as a result of the activity of saprophytic bacteria may very properly be called putrefactive alkaloids. Secondly, we have the poisonous bacterial proteids. My studies lead me to believe that of these two classes of bacterial poisons the proteids are the more frequently present in infected foods. It has been asked whether or not it is possible to induce poisonous effects by the administration of these proteids by the alimentary canal? Is it not true that they are non-diffusible, and that they would be inert if given by the mouth? There is not enough experimental evidence in our possession to enable us to answer this question with certainty, but we have good reasons for assuming that they are absorbed. In the first place, we must remember that diffusion through dead animal membranes and absorption by the living intestinal walls are by no means identical. Unchanged egg-albumin will not diffuse through a dialyser, but that it may be absorbed from the intestines has been demonstrated. Mitchell

and Reichert found that some of the proteid poisons of the venom of serpents are absorbed by the unbroken mucous membrane.

In the second place, the bacteria in the food may penetrate the intestinal walls and elaborate their chemical products in the spleen, liver, and other organs, as the bacillus of typhoid fever and other pathogenic germs do.

Since it was found that some of the bacterial proteids are destroyed by a temperature approaching that of boiling water, it has been assumed by some that this is universally true, and that cooked meat or boiled milk cannot be poisonous, or, if so, they cannot owe their poisonous properties to these proteid bodies. This is an assumption which we are at present hardly justified in making. Certainly, some of the bacterial proteids can be kept for 10 or 15 minutes at 100° C., and for a much longer period at 80° C., without being destroyed. The proteid which I have with me has been dried to a constant weight at 100° C. without any appreciable decrease in toxicity, and in solution it may be heated at 82° for half an hour with no effect. However, prolonged heat renders it inert.

There are poisonous foods in which I have been unable to find either poisonous basic or proteid bodies. I was recently called upon to investigate some mincemeat which had seriously affected a number of persons. Some of this meat was fed to cats and dogs, and invariably produced vomiting and purging. This was equally true when the meat was given either raw or cooked. Notwithstanding this positive evidence of the poisonous character of the food, I have been wholly unable to ascertain the nature of its active constituent. It has been tested by the most thorough methods for inorganic poisons, for active ptomaines, and for proteid poisons, but with wholly negative results. Furthermore, plate cultures were made, and the isolated germs, both in pure and mixed cultures, were fed to and injected into animals without effect. It is possible that the poisonous constituent was destroyed by the manipulations resorted to in the attempts to isolate it.

We now know of the existence of a poisonous base and of poisonous proteids in certain samples of cheese. However, if we expect to find that all samples of poisonous cheese are due to the presence of one and the same agent, we shall be disappointed. Indeed it is, with our present knowledge of the manner in which these poisons are formed, very unscientific for us to expect to find one poison responsible for the effects which follow the eating of different samples of poisonous cheese. We must remember that these poisons are due to a variety of species of germs, and that the chemical nature of the product is not only likely to vary with the kind of germ, but with the stage of putrefaction. Indeed, it is altogether probable that different parts of the same cheese may contain colonies of wholly distinct germs, and consequently different poisons. It certainly is a fact that one portion of a cheese may be poisonous and other portions not poisonous. I have seen samples of cheese the outer portions of which could be eaten with impunity, while the inner portions were highly poisonous, both to man and the lower animals, and Ehrhart has reported an instance of the same kind.

The method of making cheese practised in America, and in other countries so far as I know, is especially favourable for the collection of a large number and variety of saprophytic germs. The milk is brought to the factory by the farmers of the vicinity. There is no intelligent inspection of the cows, their food, drinking-water supply, or stabling. Some of the milk-cans are properly scalded and aired, while there is always the probability that others are not. The milking may be done in filthy stalls, with dirty hands, from unclean udders, and, possibly, into pails which have not received proper care. Then, I believe, that there is no country in which the standard of honesty absolutely prohibits every milkman from diluting the lacteal fluid, and sterilized water is not usually employed for this purpose. Moreover, the cheese-maker himself is not always duly appreciative of the necessity of cleanliness about the factory and in the manipulations to which the milk is subjected.

The only practical suggestion which I can make for the prevention of the manufacture of poisonous cheese is the following:—Every cheese-maker should be or should employ a practical bacteriologist, and he should thoroughly acquaint himself with the germs and moulds which are necessary to the proper formation and ripening of the cheese. Pure cultures of these germs should be added to the milk, and all others excluded. A plan like this will be adopted some time, and when it is carried out intelligently poisonous cheese will not be made, and, moreover, the flavour and digestibility of the cheese made will be greatly improved. At present the bacterial flora of the cheese which we eat is dependent wholly upon accident. It is probably well that we are not acquainted with all the varieties of microscopic vegetable life which have been gathered from the barn-yards of the milkmen and which we eat in our cheese.

The infection of milk is one of the most serious questions which can interest the student of public health, as it constitutes one of the most important factors in the causation of infantile mortality, which must be admitted to be great in every part of the civilised world. More than one-quarter of the children born in the United States die before they reach five years of age, and the same high mortality holds good, I believe, for the principal countries of Europe. A large proportion of these deaths is due, either directly or indirectly, to the diarrhœas of infancy. These diarrhœas are most prevalent among children artificially fed, and they are not due to one specific germ, but may be caused by any one of a large number of micro-organisms, as has been shown by the labours of Booker and Jeffries in America, and of Escherich and Baginsky in Germany, as well as by many others. These germs are saprophytic, and their harmful effects are due to the chemical poisons which they elaborate. Baginsky and Stadthagen have obtained from cultures of a saprophytic germ from the stool of a child with diarrhœa a poisonous base and a poisonous proteid, and I myself have isolated from pure cultures of each of three of Booker's germs a poisonous proteid.

Recognition of the fact that infected milk is a potent factor in the causation of infantile mortality has led to the sterilization of milk used in infant feeding, and while a lessened death-rate has resulted, the

medical profession in my own country, at least, has been greatly disappointed in the extent of this result. The attempted sterilization of milk has largely failed for the following reasons:—

(1.) The sterilization of milk is difficult, and, as often attempted, is not accomplished.

(2.) Even when the sterilization is efficient there is no evidence that the chemical poison is destroyed.

(3.) The food is often re-infected in the alimentary canal of the child.

While it is better to feed a healthy child with sterilized milk than with that which is infected with toxicogenic germs, it would be better still could milk which has never been infected be obtained. I am also satisfied that in the feeding of a child already suffering from summer diarrhœa, it is better to wholly withhold milk for some days than to give sterilized milk, because the latter immediately becomes re-infected in the stomach and intestines.

I cannot close this paper without emphasising the importance of the work which those who are called upon to investigate cases of food-poisoning must do. The method of procedure should be both chemical and bacteriological. A careful and thorough search for inorganic poisons should be made. The germs found should be grown under conditions as nearly identical with those under which the suspected food has existed as can be obtained. Tests should be made not only for the basic products of putrefaction, but for the bacterial proteids as well. Physiological tests upon lower animals should never be neglected.

## DISCUSSION.

**Dr. R. Blanchard** (Paris) dit:—Je désire dire quelques mots des intoxications consécutives à l'ingestion de la chair des animaux supérieurs. Ces intoxications sont désignées généralement sous le nom collectif de *botulisme*. Or, il me semble qu'on comprend sous ce nom des accidents qui différent essentiellement les uns des autres quant à leur cause originelle. Par botulisme (du mot latin *botulus*, saucisson), on a entendu tout d'abord l'empoisonnement consécutif à l'usage de saucissons avariés, corrompus, putréfiés plus ou moins complètement; cet empoisonnement porte encore parfois le nom d'*allantiasis*. Par la suite, l'acception du mot botulisme est devenue plus large, et l'on en est venu à désigner ainsi l'intoxication par une viande d'espèce quelconque. Je crois pouvoir démontrer qu'il y a là une regrettable confusion. La corruption des viandes est due, on le sait maintenant, à ce que des microbes les ont envahies et se sont nourris et multipliés à leurs dépens, produisant d'autre part des substances chimiques, les *ptomaïnes*, dont la plupart sont éminemment toxiques. Les intoxications par les viandes reconnaissent cette cause dans la majorité des cas, au moins dans nos pays; mais tous les cas ne sauraient s'expliquer de cette manière. Il existe en effet un bon nombre d'animaux dont la chair est toxique normalement, physiologiquement, en dehors de toute putréfaction et de tout état morbide. Sans parler ici des anguilles, dans le sang desquelles le professeur Mosso

a découvert une substance venimeuse, l'ichthyotoxine, je me bornerai à rappeler les intoxications par divers poissons de la mer des Antilles (*Meletta venenosa*) ou de la mer du Japon (*Diodon, Tetrodon*). Pour la melette, il semble que le principe toxique réside dans les muscles ; pour les Plectognathes désignés au Japon sous le nom de *fougou*, on sait, depuis les recherches de mon collègue, M. Rémy, que le poison siège exclusivement dans les glandes génitales, et qu'il s'y développe d'une façon surabondante au moment du frai, c'est-à-dire quand les glandes sont elles-mêmes en suractivité physiologique. Dans un cas comme dans l'autre, il s'agit donc de substances chimiques qui résultent exclusivement de l'activité physiologique des tissus : autrement dit, l'intoxication est due à l'ingestion de *leucomaïnes*.

Voilà donc deux catégories bien distinctes d'intoxications par la chair des vertébrés :

1° le *botulisme* est l'intoxication par la viande envahie par les microbes et dans laquelle ceux-ci ont déposé des *ptomaïnes*. Par définition, ce mot s'applique non seulement à l'intoxication par les viandes abattues depuis un temps plus ou moins long et déjà corrompues, mais aussi aux viandes de conserve.

2°, la *signatère* est l'intoxication par la viande fraîche, dépourvue de microbes, mais chargée de *leucomaïnes* résultant de l'activité physiologique des tissus. Je propose d'attribuer à cette catégorie d'intoxications le nom de *signatère* ou de *signatera*, nom déjà employé par les médecins espagnols des Antilles pour désigner l'empoisonnement par la melette et autres poissons vénéneux.

Au botulisme se rattachent, dans un certain sens, les intoxications produites par l'ingestion de la chair d'animaux atteints de maladies infectieuses et dont les tissus, par conséquent, sont chargés, soit de ptomaïnes élaborées par les microbes pathogènes, soit de leucomaïnes produites par les organes, en raison des mauvaises conditions physiologiques causées par la maladie. Gärtner a fait connaître un curieux cas d'intoxication par la chair d'un veau malade, chair dans laquelle il trouva le *Bacillus enteritidis*. On connaît d'autre part un bon nombre de cas analogues. Je n'insiste pas sur ce cas particulier, sur lequel je reviendrai, s'il y a lieu, au cours de la discussion sur les maladies infectieuses transmissibles des animaux à l'homme.

A la signatère se rattachent, au contraire, les intoxications par les œufs de poisson, de crustacé, de mollusques, ainsi que les légers accidents qui peuvent suivre l'ingestion de la viande des animaux surmenés. Dans les chasses à courre, les muscles du cerf fournissent un travail considérable : les produits de désassimilation sont fabriqués en surabondance et ne peuvent être évacués assez activement. Il en résulte que la chair musculaire acquiert un goût désagréable, voire même une légère toxicité.

**Dr. George Fleming, C.B.** (London), said that Prof. Blanchard had shown in a remarkably lucid and interesting manner the source of production of physiological and pathological materials, which were considered dangerous in certain foods, and the statements of Dr. Ballard and of Dr. Vaughan corroborated what had been often reported. The conditions under which dairies were kept and cheese was made were also points about which there could be no doubt as to their being in many cases most unfavourable. But were the conditions under which flesh was prepared in the slaughter-houses of only too many towns any more favourable?

They were often filthy in the extreme, and, even if they were otherwise, the localities in which they were situated were most foul. In discussing the question of meat-infection, this fact should not be lost sight of. The subject was a most important one, and its discussion should have some practical results. The natural history of the pathogenic organisms was now advanced to a stage at which some conclusions should be arrived at with regard to preventing their malignant operation. The contemptibly minute organisms were infinitely potent for mischief, and though it was necessary to study thoroughly their nature, it was still more necessary to know how to prevent their action. How was food to be protected from the changes induced by such organisms—flesh and milk and canned food? Sanitarians should deal with this practical aspect of the matter, as the public would expect to be safeguarded from danger.

**Prof. Brown, C.B.** (London), after complimenting Dr. Vaughan upon the valuable information he had communicated, wished to offer a few words in regard to the points that had been prominently brought forward. It was quite evident that there were points still undetermined which required the investigation of bacteriologists. It was abundantly quite clear that there were microbes having no special morphological character, which might undergo various changes under cultivation and produce unanticipated effects. The bacteriologist was at present somewhat hampered in regard to his cultivating medium. It was already quite well known, as the reader of the paper had said, that some organisms would only grow in the presence of air, and that others refused to grow under such conditions. It was a question whether some of the diseases were due to organisms at all.

He sympathised most fully with Dr. Vaughan's suggestion that every cheese-maker should employ a bacteriologist; and he had no doubt that the bacteriologists present would look forward to that happy time with feelings of fond anticipation. But, short of that, he could not help thinking that something should be done to employ bacteriologists in connexion with cheese-making, and to relieve them from the present unhappy condition of things. With regard to dairy produce, no one but the inexperienced in dairies could fail to be alive to the fact that while there were some model institutions in which everything was perfect, the ordinary surroundings of the dairy in the typical dairy district were most filthy, and the emanations from such dairies were easily recognised by the passers-by.

It was a misfortune that dairy products should be collected under such insanitary conditions. He agreed with Dr. Vaughan in his suggestion that something should be done to relieve them from this state of things in a country which was presumed to take an interest in sanitary science.

# Infectious Udder Diseases of the Cow in relation to Epidemic Diseases in the Human Subject.*

BY

E. Klein, M.D., F.R.S., Lecturer on General Anatomy and Physiology in the Medical School of St. Bartholomew's Hospital, London.

## Introductory.

On various occasions prior to 1885, epidemic outbreaks of diphtheria and scarlet fever amongst the consumers of particular milk had been investigated by the Medical Department of the Local Government Board, as to some of which it was possible to assert, notwithstanding the milk consumed was found to be the true vehicle of the contagium, that none of the agencies by which milk, after being taken from the cow, may become infective, could with any shadow of probability have been in operation. Of this character was the epidemic of diphtheria that occurred in Kilburn and St. John's Wood in 1878, and the epidemic of scarlatina in St. Giles and St. Pancras in 1882. In these instances, experienced medical officers, who carefully investigated all the details of the outbreaks, satisfied themselves as to the non-admissibility of any of the then recognised modes of furnishing the milk with infective power; and, as the medical officer of the Local Government Board puts it, "the "question of risk from specific fouling of milk by particular cows "suffering, whether recognised or not, from specific disease was seen to "be arising."

## I.—Scarlatina.

It was at the beginning of 1886 that a direct answer was forthcoming to the above question, as regards scarlet fever

At the end of 1885 an extensive outbreak of scarlet fever occurred in Marylebone, which Mr. Wynter Blyth traced to be due to the consumption of milk coming from a particular farm at Hendon, and to this milk only. Mr. Power further found that cases of scarlet fever occurring at the same time in other districts of the metropolis were likewise to be referred to the milk from the same farm.

With Dr. Cameron, Medical Officer of Health of the Hendon District, Mr. Power was able to exclude all and every accidental contamination of the milk of this farm from a human source. Mr. Power further showed "that only certain sections of the milk supplies within "the farm, and eventually certain cows, had to do with the infectivity "of the milk." The milk in question was proved to have possessed infective property, day after day, from about the end of November till after the third week of December.

* This paper was copiously illustrated by lantern demonstrations of the various organisms referred to.

As to the exclusion of commonly accepted sources of fouling of this milk, this is what Mr. Power states (his Report, 1885–1886, p. 15) :—

"The dairy farmer, though willing to afford, and though indeed desirous of affording us every assistance, was utterly incredulous of the presumptive evidence tending to connect disease with the milk supplied from his farm, and till a late period of the inquiry, he remained so. His cowmen were perfectly incredulous also. And truly, having regard to the facts that we first elicited, as to freedom from illness of those at the farm, and as to the peculiar care given to the sanitary affairs of the farm and its dairy, the farmer's incredulity could not but be regarded as justifiable. He had certainly done his best to avoid known conditions of danger, and had not suspected that any such condition, known or unknown, had been present on his farm. The farm was found to have had especial pains taken to render it, as the phrase is, sanitarily perfect. At the instance of one of the London retailers, with whom the farm had dealings, the place had, for several years, been the subject of special supervision by the medical officer of health of the district, my coadjutor in this inquiry, Dr. Cameron. He had seen that the West Middlesex Company's water was laid on to the farmhouse, to the dairy, and each of the several cow-sheds; he had seen especially to the wholesomeness, as regards drainage, cleanliness, ventilation, and the like, of the house, the farmyard, the cowsheds, and the dairy, securing for the last all needful appliances for effectual cleansing of dairy utensils by hot water or steam; and, month by month, he had inspected the farm premises with reference to these and similar details, for the express purpose of safeguarding the milk against contamination of any detectable kind. Further, under the same arrangement, Dr. Cameron had specially attended to the health-conditions of those employed about the farm and their children, with a view to early detection of any malady among them that might by chance injuriously affect the milk with which they had to do. He had even undertaken to observe and to report to the London retailer, by whom his services were retained, on any occurrences of infectious illness in the neighbourhood of the farm, even though it did not directly affect the families of people employed there. The farmer, too, who had consented to the exercise of this supervision over his doings, had attended to every suggestion made to him, and had taken every precaution to secure his farm and his milk against any known sanitary fault or misadventure. He had a separate shed for any sick animal, and a separate shed for the observation of newly arrived animals.

"Thus, with Dr. Cameron's aid, the point was speedily reached, at which it could provisionally be affirmed of the Hendon farm milk that, if indeed it had caused scarlatina among its consumers, it had not acquired the ability to do so in any commonly accepted way, such as through unwholesome conditions of water or drainage, or through careless handling of milk, or milk utensils by persons carrying scarlatina infection. Nor, during the long subsequent acquaintance with the farm gained in the course of this inquiry, did any reason appear for modifying this conclusion."

The next important fact that Mr. Power ascertained was that shortly before the appearance of the scarlet fever amongst the consumers of the Hendon milk, three recently-calved milch cows had been acquired about the 15th of November from a dealer in Derby, and that not until the milk of these cows was added to the milk of the rest of the farm did

scarlet fever cases make their appearance among consumers of the milk. These cows had, soon after their arrival at the farm, been affected with an ulcerative eruption on the teats and udder, and from them, during the process of milking, *i.e.*, by the milkers' hands, the disease was communicated to other cows at the farm. The medical officer of the Local Government Board, in his report for 1885–1886, pp. vi and vii, sums up the evidence concisely thus :—

"In the end, Mr. Power has demonstrated beyond reasonable doubt the dependence of the milk-scarlatina of December on a diseased condition of certain milch cows at the farm; a condition first introduced there in the previous month by some animals newly arrived from Derbyshire; and he finds strong circumstantial evidence for believing that the latter phenomena of this dependence was brought about through the extension of the diseased condition of one set of animals to another set, after the fashion of an infection."

This disease in the Hendon cows manifested itself in the following symptoms: ulcerations covered with scabs on the teats and udder; ulceration, scabs, scurfiness, and loss of hair in patches in different parts of the skin; the animals were thin and had slight cough, but showed no rise of temperature. The viscera of two animals killed showed slight pleuritis with recent fibrinous deposits and adhesions, congestion and extravasations of blood in many lobules of the lungs, discoloured softened patches in the liver-tissue, ecchymoses and petechiæ in the tissue and in the capsule of the spleen, swelling of, and extravasations of blood in, the lymphatic glands, congestion and glomerulo-nephritis of the cortex of the kidney; in some also interstitial new growth.

The chief objections* that have been raised against the inferences of Mr. Power, Dr. Cameron, and myself can be summarised as follows :—

(1.) The first cases of scarlet fever in Marylebone did not coincide in point of time with the addition of the new arrivals, *i.e.*, the 15th of November cows, to the Hendon stock. It has been contended by Professor Axe that the incubation period of scarlet fever in the human subject is from two to six days, and that therefore the London outbreak should, if the Hendon cow disease had caused it, have disclosed itself much earlier than it did; namely, about the 18th to the 21st November, instead of toward the end of that month. This objection is a notable one, for the reason that it assumes, quite gratuitously, that one or more of these cows were actually suffering from the cow disease at the date of their reception at the Hendon farm, and then goes on to ignore altogether certain obvious considerations that would, even on that assumption, readily account for the observed interval between reception of the cows at the farm and the outbreak of scarlet fever among consumers of the milk. These considerations are :—

1. Possibility that at the date of arrival of the three cows at the Hendon farm such of them as were already ill might not have

* I may be permitted to review these in detail, because the conclusions arrived at by Mr. Power, Dr. Cameron, and myself stand or fall according to whether these objections are considered incorrect or valid, and at the outset I may be allowed to remark that the nature of these objections is, as I shall show, such that those who have raised them could not have carefully read our reports on the subject.

yet reached a stage of their malady at which their milk secretion could contain material of infection.

2. Possibility that on their arrival such of them as were ill might have advanced so far toward recovery as to have passed that stage of illness to which infection-material from their tissues could be discharged in their milk, in which cases the milk from the farm need not have been infected until a companion cow or cows had in due course caught the malady and arrived at that stage of it in which infection became mingled with such companion cow's milk.
3. Possibility that for several days after reception of these three cows at the Hendon farm all milk furnished by them might (as was the custom there with milk of new cows) have gone to make butter.

(2.) The next point urged was this (see Professor Axe in Professor Brown's Report to the Agricultural Department*) :—As stated above, on the 15th November cows had been bought from the Derbyshire dealer; these cows, it has been shown, developed an ulcerative eruption on the teats and udder, which they communicated to other cows in the Hendon farm. Now it has been stated by Professor Axe that the three cows in question were part of a lot of 30 bought in Derby Market on October 16th and 30th, and that same dealer had sold, from this lot of 30, other cows also affected with an ulcerative eruption on the teats and udder to other and different localities, in which localities also ulcerative eruption had spread to the cows of the farm, but that no scarlet fever had been produced by the milk of these farms.

In the first place, this information as to that Derbyshire dealer having procured 27 other cows at the *same time*, and from the *same batch* from which the 15th November cows were part, was given to Professor Axe by that dealer many months after the Hendon inquiry had terminated; at the time that the Hendon inquiry was proceeding the dealer, who was believed to have purchased these cows in Derby Market, and who sold them to the Hendon farmer, resolutely refused all information whatever. Such evidence, therefore, given by that dealer to Professor Axe is, for purposes of a scientific argument, absolutely untrustworthy, particularly if it be remembered that the dealer in giving information to Professor Axe was only doing what he, from the point of view of a dealer, no doubt thought the most advantageous thing for his business. But, moreover, as a matter of fact, the three cows in question which were selected by the Hendon farmer from among the stock at this dealer's depôt, were, the farmer said, newly arrived from Derby when he chose them there on 14th November, and accordingly had no doubt been purchased in Derby Market at a date subsequent to the 30th October. So far, therefore, as the story of the distribution by this dealer of other cows (besides the three in question) affected with udder eruption is correct, it is suggestive of distribution of more than

* Report on eruptive disease of the teats and udder of cows in relation to scarlet fever, 1888.

one sort of cow malady to persons purchasing cows of him in October and November 1885. But there seems to me another point worth remembering. I had the opportunity within this year of visiting a dealer's place in Surrey, and I there saw a number of cows aggregated in a shed; they had all been bought at Reading, market some a few days before, others the week before; they were all waiting to be sold; the dealer knew nothing more about these cows than that he bought them at Reading; where they originally came from did not in the least concern him; "his business was to sell them, they are here to-day, they are sold "to-morrow." I presume that this really represents the sum total of a dealer's knowledge and interest in the cows that he buys and sells.

(3.) The next objection urged by Professor Axe is of an altogether different character, it is this: Granted that the milk of the Hendon farm did prove capable of giving scarlet fever to the consumers, it does not necessarily follow, he contends, that the milk received its infective power from the cows. On the contrary, Professor Axe has "gathered" that a boy living at "The Mead," about three-quarters of a mile distant from the farm, and who, being "on intimate terms" with the farm bailiff, visited the Hendon farm, came from a house in "The Mead" in which a case of scarlet fever had occurred at about the same time as the cases in Marylebone. Professor Brown, in his Report to the Agricultural Department of the Privy Council, accepts this "discovery" of Professor Axe of a suspicious boy as conclusive proof that the Hendon cows had been accused unjustly of being the primary cause of the scarlatina.

Dr. Thin, in his address to the British Medical Association, August, 1887, said:—

"Two men, I am assured, who were engaged as milkers at Panter's Farm, lived in 'The Mead,' walking to and from their work, a distance not over half a mile. There is not the slightest proof that these men conveyed the scarlatinal infection from 'The Mead' to Panter's Farm, but it is quite conceivable that they may have done so; the very fact that 'The Mead' contains so many laundries, that scarlet fever existed at Child's Hill, and that there was daily communication between 'The Mead' and the milking-shed, justifies a suspicion, or at all events affords a possible explanation of how the milk became infected."

I am referring to this hearsay evidence of Dr. Thin chiefly because I saw it quoted at the time in a Vienna medical paper. But in the light of the positive evidence on this very subject, by Mr. Power and Dr. Cameron, the health officer of the district, specially retained to superintend the farm, its employees and their families (*see* pages 131–2), Dr. Thin's thesis, of two men being allowed to carry the contagium of scarlet fever to the Hendon farm, deserves no serious consideration.*

It is not much different with the "boy" "discovered" by Professor Axe. Dr. Cameron, in his Report to his Board, distinctly and in

* As a matter of fact, Dr. Cameron, in his report for 1885, states, page 19:—"In the last week of November two mild cases of scarlet fever came to my knowledge in 'the Mead,' Child's Hill. Both were carefully isolated, and disinfectants "supplied . . . . The fever did not spread beyond either of these houses."

detail describes the cases of scarlatina at Child's Hill, so that he and Mr. Power were perfectly aware of these cases, but they could not, after the most diligent search, find that any communication, directly or indirectly, had taken place between these cases and the Hendon farm.

I want my audience to consider for a moment what the actual conditions of the milk at the farm were. The milk was proved to have possessed infective power from day to day for over three weeks, and therefore the cause for that infectivity must have recurred day after day for at least three weeks. Granted even that the boy discovered by Professor Axe did come one or twice—it is not alleged that he came often—to the farm from a house which contained a scarlet fever case (isolated in one of its upper rooms), and granted also that the boy was the vehicle for the contagium to the farm, this could have only occurred once or twice; therefore, even granting this assumption of the boy having visited the farm, the further assumption that the milk in a well-regulated farm, worked on the most sanitary principles as to water, employés, utensils, &c., as the Hendon farm is acknowledged to have been, could have thus acquired and retained infectivity for over three weeks is absolutely incredible. Supposing this boy had purposely carried a handful of contagium to the farm and had access to the milk, and had deposited it in the milk on a particular day, one could understand how that particular sample of milk could have given scarlet fever; but that milk, delivered three, four, or more days afterwards, that is to say, milk that had not been even secreted by the cow at the date of the boy's visit, could give scarlatina, is, on the face of it, absurd. One might further ask how could the boy have infected the milk, since the cows were on this, as in other farms, milked not long after midnight, and the milk is sent off to the retailer in the early morning; the boy is obviously not likely to have paid his visits to the sheds of the farm before dawn, nor indeed by the time the milk was sent off, but some hours later. After Professor Axe's Report was published, detailed and further inquiry was made about that boy, and "as the result of definite local "inquiry at the lad's home and elsewhere, it has also been 'gathered' that "the story is practically, if not wholly, mythical." (*Practitioner*, March 1889.)

It ought not to be omitted, before leaving this theory of Professor Axe, to point out that in the same volume of the Agricultural Department in which Professor Axe's Report is published, there is an important paper by Dr. Hime. In this Dr. Hime shows that even given a case of scarlet fever at a particular dairy farm itself, the milk of this farm did not convey scarlet fever.

As an exhaustive summary in criticising Professor Brown's and Professor Axe's Reports on the Hendon outbreak, I append here an extract from the *Practitioner*, March 1889. I am anxious to bring this subject before the veterinary profession and our continental colleagues, since it is very probable that they have only had before them Professor Brown's volume, and may not have "heard the other side," as stated in the *Practitioner*, pp. 225-234.

"Scarlatina in several different quarters of London was admittedly traced to the use of milk from a Hendon farm. The infection commenced

shortly after the reception at this dairy-farm of certain recently-calved cows newly arrived from Derby. Customers of the several retail milk businesses in London were or were not attacked, according as the milk they got came or did not come from sheds at Hendon, containing either the Derby cows or cows which had become infected from them; and these Derby cows and the others infected by them were suffering from a specific disease which is now provisionally call the Hendon disease, and which, amongst other symptoms, has associated with it an eruptive affection of the udder and teats. So long as and wherever the milk from these cows was used, scarlatina was produced; when it was discontinued the disease abated, and this although milk from other cows at the same dairy farm not affected with the Hendon disease remained in use. And as the result of prolonged inquiry by both Mr. Power and Dr. Cameron, the local medical officer of health, it was found that the milk could not have acquired the ability to convey scarlatina 'in any commonly accepted 'way, such as through unwholesome conditions of water or drainage, or 'through careless handling of milk or milk utensils by persons carrying 'scarlatina infection.' And, as the outcome of an inquiry, the report on which, to quote Dr. Buchanan, must 'be studied before the exactness of 'his observations and the validity of his inferences can be duly ap-'prehended," Mr. Power had the conclusion forced upon him that the milk scarlatina in question was demonstrated to be due to a diseased condition of certain milch cows at the implicated farm.

"Discussing these conclusions, Professor Brown at the outset refers to Mr. Power's work in this connexion as if it were a solitary observation. This is distinctly misleading, for the subject had for some years been under investigation, and Dr. Buchanan, in referring in his reports on the proceedings of the Medical Department for 1882 to the distribution of infectious diseases through communities by the agency of milk, wrote: 'In such outbreaks, contamination of the milk by the material or 'specific disease derived from some antecedent case in the human subject 'has been of necessity the first explanation that has offered itself for 'acceptance, and in many such outbreaks this explanation has appeared 'valid and sufficient. But in outbreaks of scarlatina more especially 'such a hypothesis of the manner in which the milk may have obtained 'its specific quality has not always accorded with the observed facts, and 'not unfrequently it has appeared, during recent epidemics, that for all 'we can affirm to the contrary, the endowment of the milk with harmful 'attributes may be an affair of the very system of the animal furnishing 'the milk.' And, after discussing the details of an actual outbreak of milk scarlatina, he proceeds:—'Thus, a hypothesis that the milk actually 'possessed, at the moment of entering the pail, the power which it 'assuredly had, of producing scarlatina in the human subject, forced 'itself upon consideration; and although, indeed, the thing was beyond 'customary experience, such a hypothesis was seen to accord with the 'observed facts of the outbreak.'

"And in the volume in which Mr. Power's report on the Hendon occurrences was included, Dr. Buchanan takes care to remind his reader that the view put forward was no new one. Recalling the circumstance that infection of milk by human agency had generally been held to afford the 'readiest explanation' of the facts of milk scarlatina epidemics, he goes on to observe that 'as successive epidemics have occurred and have 'been found capable of more exact study, distrust of this explanation has 'arisen, and the means by which the milk receives infective properties 'had come to be regarded as unknown.' Indeed, it is shown that ever

since 1878 'the question of risk from specific fouling of milk by par-' ticular cows suffering, whether recognised or not, from specific disease ' was seen to be arising;' and further, that, as regards milk scarlatina, special suspicion attached to cows suffering from certain ailments which followed on recent calving. Thus, it is evident that the question of the communication of infectious diseases to man through the agency of milk, apart from any prior infection of the milk by means of a human agency, had been for many years under consideration.

Professor Brown next states that, 'early in the inquiry, Mr. Power ' had reached the point of excluding all ordinary sources of infection of ' the milk,' and, in view of this implied hasty action taken under certain specified 'circumstances,' he taxes Mr. Power with 'neglect' for not carrying his inquiry to two other farms, which, in addition to the Hendon one, sent milk into those portions of London where the Hendon milk supply had led to the distribution of scarlatina. This 'neglect' Professor Brown regrets, 'because it leaves it open to anybody to suggest that the ' result of the investigation might have absolved the Hendon milk from ' the charge of being infective, and at the same time have accounted for ' the curious fact that it did not appear to be injurious until it was taken ' some distance from its place of origin.'

"It may at once be seen that Professor Brown himself in no way absolves the Hendon milk either from being infective or from having received its infectiveness at Hendon, for, as will presently be learnt, he assigns a local cause for the infection, operating at the farm, and of which he says that it may be 'reasonably accepted.' And as to the 'curious fact' of the milk not appearing injurious except when at a distance from its place of origin, Professor Brown has forgotten one of the most crucial pieces of evidence as to the infectiveness of this milk, which Mr. Power adduced as 'a most instructive but pitiful experiment' actually going on at Hendon.' The facts referred to are these: The dairy farmer, having his milk returned on his hands, gave order that the portion of his produce which he regarded as being under suspicion should be used either for pig-feeding or be thrown into a pit freshly dug in his fields. But certain poor people living in houses at Child's Hill, 'and elsewhere near the farm,' pleaded with some of the cowmen that they might have some of the discarded milk, and within about a week scarlatina assumed 'the pro-' portions of a little epidemic' amongst the families to whom the milk had been given, whereas 'it attacked no family to whom it was not supplied.' There is also recorded evidence that a daughter of a cowman at the farm took scarlatina 'almost simultaneously' with the date at which the London consumers of milk from a newly infected shed began to suffer. And again, Mr. Power reports how 'a friend of the farmer, who had by ' favour been supplied with the milk of a single cow, had had his family ' very heavily stricken *at Hendon* about the end of the first week in ' December.'

"Then, as to Mr. Power having 'early in the inquiry' excluded all ordinary sources of infection, Professor Brown himself shows that no reference is made in the report to any such exclusion, until nine closely printed pages out of the twelve of which Mr. Power's report is made up have been occupied in discussing the epidemic and the sources of the infection giving rise to it; and he ignores that which is so apparent to the ordinary reader, namely, the obvious fact that Mr. Power, with the help of the local health officer, and with that of the owner of the dairy farm and his employés, was during several weeks and practically day by

day investigating this very point; and that the utmost care was exercised by all concerned, not only to detect any ordinary source of infection, but also to keep every possibility of it off the farm premises. It is quite true that in face of overwhelming evidence in a contrary direction, this view as to the absence of any ordinary mode of infection was 'provisionally' reached at an earlier stage; but the whole body of the report gives indications that the possibility of a human source of infection was throughout kept in view, and this not only by Mr. Power and Dr. Cameron, but by interested parties, who remained 'utterly incredulous' about any source of mischief in the cows themselves.

"The 'circumstances,' too, which led Mr. Power to regard ordinary sources of infection as having been eliminated, were also by no means limited to any mere consideration of contagion direct or indirect from a prior attack of scarlatina, and it is noteworthy that Professor Brown's quotation as to this begins in the middle only of the paragraph in which the conclusion referred to is arrived at. The 'circumstances' were multifarious, and their relations with the events investigated are discussed at length in Mr. Power's report. Above all, they led by a chain of evidence which must be regarded as at least very circumstantial, to connect the disease amongst the London milk drinkers with certain definite cows at Hendon. The paragraph as it stands is as follows:—

"'We had thus reached the point of excluding external scarlatina, of associating the importation of particular cows into the Hendon farm with the presence of scarlatina in London districts, and of connecting by a series of parallel events the milk furnished by those cows and by related cows, with the peculiarities of scarlatina prevalence amongst consumers of the Hendon farmer's milk. Under these circumstances, it was not judged necessary to go beyond the Hendon farm, and to inquire at the two other farms that also sent milk into the London districts of South Marylebone, Hampstead, and St. Pancras, in search of the cause of scarlatina in those districts.* Henceforward, until anything to the contrary should appear, an influence competent to produce scarlatina among the consumers of the milk was held to have operated from those cows which were received into the Hendon farm on November 15th, and the further concern of the inquiry was the nature of such influence.'

"And as to the need for inquiry into milk from other farms, it must be remembered that Mr. Power had already reported, on the authority of Mr. Wynter Blyth, the Marylebone medical officer of health, that 'customers supplied with Hendon milk, *and Hendon milk alone*,† had 'suffered scarlatina;' and also that Professor Brown himself, in his earlier report of 1887, had admitted, on what appeared to him then to constitute 'unquestionable evidence, that the outbreaks in Marylebone 'and other districts were traceable to milk obtained from a dairy farm 'at Hendon.' The grounds for Professor Brown's altered attitude a year later, to the effect that the Hendon milk might have been 'absolved,' are nowhere apparent in his report, neither does he give any good reason for taxing Mr. Power with 'neglect.'

---

* But even here Mr. Power is only speaking of the absence of exhaustive investigation at those other farms, such as was made at Hendon, for we learn from Dr. Cameron's Annual Report for 1885 that such inquiry was made, and an inquiry at a farm at Swindon is especially referred to in this connexion.

† The italics are in the original.

"At this stage it will be convenient to pass on to an alleged discovery made by Professor Axe, which evidently gives the special tone and character to Professor Brown's report. According to that report, Professor Brown states that Professor Axe, (finding 'reason to doubt* the alleged relation between the Hendon outbreak and the outbreaks of scarlatina,' proceeded to other inquiries, '*and he at length ascertained*' that scarlatina existed in Hendon during several months of the year 1885, particularly in the neighbourhood of Child's Hill; and at the time when the disease appeared in London it was known to exist in 'The Mead.' And the further adds, that of fourteen men employed at the dairy farm six resided in 'The Mead,' and passed backwards and forwards daily between the sheds and '*the fever district.*'

"Now, as to this discovery by Professor Axe, it may at once be stated that the facts were in no way new, that they were perfectly well known by those investigating the Hendon occurrences at the time of the outbreak; and, furthermore, that details are given of them in Dr. Cameron's Annual Report for 1885. And, in this connection, it is worth comparing the terms in which Professor Axe makes the announcement in his second report, not issued till November 1888, with those employed by Dr. Cameron in his 1885 report, issued at the beginning of 1886.

| "Dr. Cameron's Report for 1885. | "Professor Axe's Report issued by Professor Brown in 1888. |
|---|---|
| "In the last week of November two mild cases of scarlet fever came to my knowledge in "The Mead," Child's Hill. Both were carefully isolated, and disinfectants supplied. As the district is full of laundries, it is possible that the disease was introduced from London; there was no other evident *source* for it. *The fever did not spread beyond either of these houses.* | "In the last week of November two mild cases of scarlet fever came to my knowledge in "The Mead," Child's Hill. Both were carefully isolated, and disinfectants supplied. As the district is full of laundries, it is possible that the disease was introduced from London; there was no other evident *cause* for it. |

"In other words, Professor Axe is reported in 1888 to have 'at length ascertained' that which Dr. Cameron had reported on early in 1886, and his discovery is (apart from an obvious error and a highly important omission, both of which we have italicised) couched in precisely the same terms as those used by Dr. Cameron.

"Now, it is quite obvious that Professor Axe not only forgot to state that he was quoting Dr. Cameron, but also to put the quoted passage in inverted commas; and Professor Brown, being thus accidentally misled as to the authorship of the passage, was able to announce a new discovery, which must, according to the sequel in his report, have led to a different conclusion as to the concern of the cows with the infection of the Hendon milk, had it only been known before. And since Professor Axe left out the concluding words, to the effect that no spread took place beyond the two houses affected, Professor Brown doubtless felt himself justified in

* Professor Axe's own statement really goes much beyond the expression of a doubt, for he found, says he, "it impossible to reconcile" his own "facts" with Mr. Power's "assumption." The italics are ours, and the point is referred to later on.

endorsing Professor Axe when he speaks of the locality as 'the fever district.'

"And now as to the bearing of these old facts, brought up anew, on the milk scarlatina. Professor Brown evidently has two theories. One is that six men who worked in the dairy farm passed daily through a street where, at 600 yards distance from the farm, there were two isolated cases of scarlatina; and his reference to the fact as a discovery, as also his characterising the street as a 'fever district,' give an obvious indication of his views with regard to the circumstance in its possible relation to the milk infection.

"On this point the reader need not be long detained, for it is perfectly well known that passage through a locality where there are two cases of scarlatina does not affect the passers-by, and thus enable them, either in their clothing or otherwise, to carry the scarlatina infection for a distance of 600 yards. (Dr. Cameron reported it to be nearly three-quarters of a mile.) And even Professor Axe minimises the danger by explaining, as regards one of these two solitary attacks, that it occurred in a house standing at 'some distance from the road.' Besides which, since the milk was found to be infective day by day, and apparently at both the morning and the afternoon deliveries, Professor Brown's assumption would require the infection of these dairymen to have been maintained for weeks, and to have been potent for conveyance of the scarlatina poison to the milk both morning and afternoon. To all who knew anything about scarlatina and its mode of spread, such a notion is far-fetched in the extreme. Were such an occurrence likely to take place once, to say nothing of twice a day, for several weeks, no member of a population of any ordinary town or village, who must perforce run the risk of passing through localities far more dangerously circumstanced than this one in 'The Mead' ought ever to enter a dairy.

"The next theory relates to a family named Potter. A girl of this name was one of the two solitary November cases of scarlatina in 'The Mead.' She had a brother, who, as Professor Axe 'gathered' from his father, had received encouragement to visit the dairy; he, a little boy of twelve years only, being 'on intimate terms' with the bailiff; indeed much of his time is said to have been spent in such visits. With regard to this boy not a word is said as to date of visit, without which any inference is of course worthless, and this is the more significant because we learn at the same time that the lad's father, 'having heard of the cow disease and of its consequences,' also went to the farm, and this whilst 'conditions of infection still prevailed in his house.' The father's visits were therefore paid after the London milk scarlatina outbreak had occurred, and hence they can have had no bearing upon it. And further, Professor Axe himself announces that which was perfectly well-known at a much earlier date to Mr. Power and Dr. Cameron, namely, the fact that the scarlatina in Potter's daughter and the other patient in 'The Mead' occurred 'at the time when the disease appeared in London,' but he appears not to have apprehended the significance of this simultaneousness, which should only have sufficed to suggest that 'The Mead' cases, like the London cases, were due to the same infection, and not that one was the cause of the other. Notwithstanding all this, however, much is made by Professor Brown of this alleged discovery, and he repeats Professor Axe's conclusion: 'Here then we have a human source of scarlatina.'

"But to return to the dateless visits of the boy. It has been shown by Mr. Power that the beginnings, intermissions, recrudescences, and

sequences of incidence of scarlatina on the London customers were clearly related to parallel receptions in and transferences to different and varying sheds at the Hendon farm, of the Derby cows or of other cows becoming infected from them. So that this little lad, if he was the cause of the milk infection, must just have managed to contrive the disposal of the contagium he carried about with him so that it should reach just those milk pails coming from the varying sheds which contained these special cows, and that it should also studiously be kept from any others; and he must further have succeeded in thus transmitting, with the necessary discrimination, the scarlatina poison day by day for a number of weeks.

"And again, if there is one thing clear about this outbreak, it is that the morning milk supply was potent for causing scarlatina in those that consumed it. Now everyone who knows anything of the work which goes on in these country dairies whence milk is conveyed by road to London, will be aware of the fact that the dairy processes begin at about 2 a.m., and are practically over by 6 a.m. It is quite true that the darkness of these winter nights may have aided in covering this little boy's nightly truancy from his bed and from his home, and it is also possible that in this way his movements may have escaped those about the farm, including his 'intimate friend,' the bailiff, and others who were on the watch to discover any possible source of the scarlatina; but it is hardly credible that practices which must necessarily have been maintained day by day, and probably twice a day, should have been altogether unknown to those who were responsible for the management of the farm at this critical period, as also to Mr. Power and Dr. Cameron, both of whom knew all the circumstances as to scarlatina in his home.

"Professor Axe, in dealing with the occurrences related, after all only urges that the theory of infection by a human agency through the media he has implied 'is not a more remote assumption than that which 'ascribes the outbreak to some hitherto unrecognised condition of the 'cow'; but Professor Brown is not inclined merely to put it thus on a level with a theory which he practically dismisses as incredible, and hence, in summarising this part of his subject, he asks: 'If Mr. Power 'had been aware . . . . . that some of the milkers at Hendon 'lived in an adjacent place where scarlatina existed, and were in the habit 'of passing daily between the fever district and the Hendon cowsheds, and 'also that two persons who lived in a house with a scarlatina patient 'visited the cowsheds, one of them spending a considerable part of his 'time about the sheds, would he have been quite so certain as he was that 'the milk of the Hendon cows had not acquired the power to cause 'scarlatina, in any commonly accepted way, as by the agency of persons 'carrying scarlatina infection, or if he had known and stated all the facts 'which have since transpired, and had still maintained the same conclusion, 'would it have been accepted, as it has been, by many members of the 'medical profession as amounting to mathematical proof?' The answer to much of this has already been supplied, but it should be further pointed out that incidents, which Professor Axe at this point brings forward as amounting only to an assumption, are on the same page transformed into actual facts, for the lad Potter is now (if there is any meaning in the above quotation) definititely stated to have actually 'visited the cowsheds,' and to have spent 'a considerable part of his time about them.' It will be well here to recall the circumstance that all this is based on a theory which Professor Axe only "gathered" from the lad's father and

the incident may fittingly be closed by stating that, as the result of definite local inquiry at the lad's home and elsewhere, it has also been 'gathered' that the story is practically, if not wholly, mythical. * The utter improbability of the inferences deduced from it, which certainly could not, in any case, be regarded as amounting to proof, need therefore no longer concern the critical reader."

(4.) The last point which has been urged against us is by Professor Crookshank, viz., that an eruptive disease in cows investigated about the second half of November of 1887 at certain Wiltshire farms, was the same as the Hendon disease, but that the milk of these Wiltshire cows did not give scarlet fever. Professor Crookshank maintains that he has proved that the Wiltshire disease was cow-pox, and he infers that the similarity between this malady and the Hendon disease is so striking that he does not hesitate to affirm that the Hendon disease was also cow-pox. Now, whether the Wiltshire disease was cow-pox or not, I do not think it possible to say. From the evidence furnished by Professor Crookshank it is possible but by no means certain that it was cow-pox, because the evidence was in several important respects insufficient, as I have pointed out in detail in my report in 1888–1889. I say it is possible, because in the summer of 1888 I had the opportunity to investigate an eruptive cow disease in the adjoining county, viz., Gloucestershire, which I have fully proved to have been cow-pox; and it is possible, but by no means certain, that the disease in the Wiltshire cows, six months previously, was cow-pox. In both instances a considerable number of milkers had on their hand or face contracted the eruption, which in its aspect and course strongly resembled vaccinia. In the case of the Gloucestershire disease, with lymph from the vesicle on a milker's hand, calves were inoculated, they developed vaccinia and the lymph of these latter was used for vaccination of infants' arms, and the result was true vaccinia.

The Wiltshire disease had the following features in common with the Hendon disease. In both, the udder and teats showed an eruption which commenced with red raised papules, the skin much injected, the papules enlarged and became vesicles, then pustules, the enlargement still continuing and the redness abating. The pustules became covered with brown crusts, which, when removed by the milker's hand or otherwise, exposed a bleeding sore. The crusts thickened and ultimately became loose, leaving a contracting healing sore; when completely healed a slightly raised reddish scar was noticeable.

But both in the duration and character of this eruption the following well marked differences could be noticed between the Hendon eruption, as I saw it at Hendon, and the Wiltshire disease, as I saw it on a cow

---

* Since the above was written, a review of Mr. Power's and Professor Brown's reports has appeared in the *Sanitary Annual* (Williams and Norgate, London). As to this incident, it is pointed out that "on inquiry" . . . . . it appeared that a period of three years, more or less, intervened before Professor Axe interviewed the Potter family, and it is added that the bailiff "denies that the boy was ever in the cowsheds."

sent to me from Wiltshire by Professor Brown, and subsequently on a cow from Kingston:—

(*a.*) The eruption in the Hendon disease passed more rapidly through all its stages than the Wiltshire disease; the crusts in the latter remaining unaltered for weeks, whereas in several of the Hendon cows which I saw, already after ten days to a fortnight since their first appearance the crusts became loose and fell off.

(*b.*) The eruption on the teats and udder in the Hendon disease were smaller, more limited, and better defined in outline; in the Wiltshire disease the crusts and sores were extensive and irregular in shape and outline.

(*c.*) The sores in the Hendon disease had a more markedly indurated base than in the Wiltshire disease, besides the sore in the stage of the crust seemed always more distinctly raised, whereas in the Wiltshire disease the base was less infiltrated and the sore flatter.

(*d.*) In the Hendon disease, besides the eruption with crusts on the teats and udder, there were always present in other parts of the skin, particularly the back, sores with crusts, in many places the hair had fallen off and the epidermis desquamating.

(*e.*) In the Wiltshire disease infection of the milker's skin, generally the hand, with the development of an eruption resembling vaccinia, was of common occurrence; the same was the case in Kingston (see my Report, 1888-1889) and in the Gloucestershire (cow-pox) disease (see my Report, 1888-1889). But in the Hendon cow disease, though the number of cows affected with the eruption and the number of milkers (fourteen) was considerable, there was not one single instance at the Hendon farm of a milker having been infected on his skin during this outbreak. If this had been the case it would unquestionably have become known to Dr. Cameron and at the farm. This last point I consider of fundamental importance; it is a character which perhaps of all others is the best and easiest ascertained, and I should go so far as to say, that when in any dairy farm an ulcerative disease on the teats or udder of the cows appears which is of a contagious nature (viz., which is carried from cow to cow by the hands of the milker), but is not communicated to the milker's skin itself, this is not the Wiltshire disease, nor is it cow-pox.

(*f.*) In the Wiltshire and Kingston cows affected with the eruption, the post-mortem showed no disease of any kind in the viscera; in the Hendon disease, on the contrary, the viscera of two cows that were examined were found distinctly affected, and amongst these the lungs and pleura deserve the first place (*see* page 132); the disease could already be diagnosed during life by the cough and by the animals becoming gradually thinner.

Before leaving the Hendon disease, it is of interest to mention that between Christmas 1886 and January 1887 an extensive epidemic of scarlatina occurred in Wimbledon and Merton, which has been proved by Mr. C. H. Cooper of Wimbledon to have been due to milk coming from a particular dairy, and further he demonstrated that human fouling of the milk was excluded. Mr. Power has also reported on this epidemic in the Report of the Medical Officer of the Local Government Board for 1886. Mr. Power states:—

"The farmer had, it appeared, immediately he found his milk unsaleable at Wimbledon or elsewhere, taken steps towards getting rid of his cows, for he could not, as he said, under the circumstances afford to continue feeding them. Accordingly he had all his cows examined and their healthiness certified to under veterinary authority, with a view to their immediate sale; and by the time I visited the farm his whole herd of 41 cows had been sold, all but 24 of them having been already removed from his premises. Of those remaining some few appeared to be recovering from affection of the skin and udder, very similar to the malady reported on by Dr. Klein as having occurred among certain cows at Hendon." (Page 329.)

A further instructive case of an eruptive contagious disease of the cow connected with milk scarlatina occurred in 1888 at Camberwell. That this eruptive disease was of a distinctly contagious nature, though it was entirely overlooked by the inspecting veterinary surgeon, I have conclusively proved by inoculation into calves, carried on from calf to calf for several generations (see my Report, 1888–1889).

It would carry me too far for the object I have in view with this paper and for the purpose of this Section, if I entered further into several important details connected with this subject; thus, the micro-organism which I isolated from the Hendon cows and from cases of human scarlatina I still firmly maintain to be the micro-organism of scarlatina, viz., the streptococcus scarlatinæ, since with it, by subcutaneous inoculation into milch cows, I have produced the same eruptive disease on the teats and udder, and the same visceral disease as was present in the Hendon cows; further, the inoculation experiments which were carried out in numerous calves with the Hendon disease, Wiltshire disease, Kingston disease, Camberwell disease, and Gloucestershire disease, prove conclusively that the Hendon and Camberwell disease were different from the Wiltshire, Kingston, and Gloucestershire disease. As to these matters I must refer the reader to the numerous experiments and illustrations given in my papers in the reports of the medical officer of the Local Government Board, 1885–1889.

### II.—Edinburgh Disease.

I now come to another eruptive disease in milch cows, the milk of which had been shown by Dr. Cotterill to have produced in an institution in Edinburgh a febrile sore throat, which appeared to be neither scarlet fever nor diphtheria. Dr. Cotterill has shown that the source of infection in the children was to be found in the milk supply of a particular dairy, in which cows had an eruptive disease on the teats

and udders. The use of the milk from a specially separated cow that had contracted the teat disease had produced in a child this throat affection. "Between October 10th and October 20th there were four "diseased cows in the suspected dairy, and 60 cases of sore throat in the "institution. Then occurred an interval, and no fresh attacks of sore "throat took place until between November 7th and 12th, when 25 more "cases of sore throat occurred. Four more cows were also attacked "between October 24th and November 12th. The sore throat epidemic "then came to an end, but eight additional cows fell ill at intervals, "one each on separate days between November 18th and December 24th." The cessation of the sore throat cases after November 12th was due to the fact that Dr. Cotterill, having come to this conclusion as to the source of infection, "suspended" the milk supply, and the "sore throat epidemic quickly disappeared." But the milk-supply was recommenced, and sore throats of a similar character appeared again. After this all the milk was boiled before use, and the epidemic . . . . yielded.

So that here was a definite proof as to this milk coming from cows affected with a contagious eruptive disease on the teats and udder being the cause of the throat affection of children consuming it. Professor MacFadyean, who investigated the cow malady, came to the conclusion that the eruption observed by him in these cows "differed in every important respect from that of true cow-pox." (See Professor Brown's Report for 1888). I had the opportunity of observing and examining such a cow affected with the Edinburgh disease, and I can fully confirm Professor MacFadyean's conclusion; the sores and scabs in their short development and duration, in their smaller and well-defined aspect, and as regards the induration of the skin underneath, correspond with the Hendon disease to a large extent, but differed markedly from the Wiltshire disease. Besides, there was distinct visceral disease in the Edinburgh cows (see my Report, 1887–1888, p. 225); the lungs were much inflamed, the lympth glands showed extravasations of blood and the liver showed numerous hæmorrhagic patches. The experiments of cutaneous inoculation carried out on calves with crusts of the sores of the teats and udder of the cow produced a definite and well characterised eruption, differing fundamentally from vaccinia.

### III.—Diphtheria.

"Dissemination of diphtheria* by milk is now in this country so "generally accepted, and has, moreover, unhappily, been so often demon-"strated, that the fact of such dessimination of the disease needs not to "be insisted on. Since discovery of this fact, however, ten or a dozen "years ago, the means by which milk can acquire ability to convey "diphtheria has remained matter of doubt and even of controversy. But as "time has gone on it has become more and more evident that satisfactory "explanation of milk diphtheria outbreaks through infection of the milk

* Further Report on the Etiology of Diphtheria, report of the Medical Officer of the Local Government Board, 1889–1890, p. 167.

" by some antecedent human case of diphtheria is seldom forthcoming." Among more recent papers on this subject is one in the 16th report of the medical officer of the Local Government Board, by Mr. Power, respecting a milk diphtheria outbreak in 1886 at York town and Camberley, which lends itself conveniently in illustration of the difficulties met with in these cases in accounting for infectiousness of the milk. At York Town and Camberley the ability of the implicated milk to cause diphtheria in the human subject was pronounced and persistent, day by day, from the 8th to the 18th October. Nevertheless, no human source of infection of the milk could be detected, and the main outcome of inquiry had to be stated in these words :—

" The milk which did the mischief had a like quantity of infectious-
" ness on each of four several milk walks, and has to be regarded as having
" become infected before leaving the farm where it was produced."
. . . . . " The method of the infection of the milk has not, in
" the present state of knowledge on this subject, been demonstrable."

" This unsatisfactory conclusion was not, however, arrived at without
" due inquiry as to means whereby the milk might have become infected.
" After excluding as non-operative at the farm in question, one after
" another of the commonly accepted means of milk infection, the report
" goes on to consider, in provisional explanation of the observed facts,
" certain hypotheses, each of which regarded the cow herself as concerned
" in transmission of diphtheria infection." (See my Report, 1889-1890, p. 167, and passim.)

The cows of the dairy whence the milk was derived had been examined on the 13th of October, and had been certified as in good health, but it ought to be added " that at the beginning of November
" one cow at the farm which had suffered with 'chapped teats,' had
" still at the site of a chap, a scab or crust not unlike those which, at a
" later stage of this malady, had been observed to replace ulcers on the
" udders of certain Hendon cows."

In Barking, an outbreak of diphtheria towards the autumn of 1888 investigated by Dr. Mason, was likewise brought in relation to milk coming from a particular dairy; here cows were affected with an eruption on the teats, followed by udder sores and crusts. And lastly, an epidemic of diphtheria occurred at Croydon towards the end of 1890, as to which Dr. Philpot proved its dependence on the distribution of milk coming from a particular dairy farm, while no fouling of the implicated milk from a case of human diphtheria could be discovered. But what was very striking was the existence at that dairy farm of an eruptive disease of the teats and udder spreading amongst the cows. There were sores on the teats, few on the udder, rounded and raised, and covered with brown crusts; the eruption passed through a rapid course, since the crusts a few days after they appeared became loose and detached, and the sores quickly healed. None of the milkers at this farm had contracted the eruption on their hands.

I have made experiments on milch cows with the diphtheria bacillus derived from the diphtheritic membrane of human throat diphtheria, which appear to me to throw a good deal of light on the above outbreaks of diphtheria.

Two milch cows* were inoculated with a broth culture of the diphtheria bacillus derived from human diphtheria. In each case a Pravaz syringeful was injected into the subcutaneous and muscular tissue of the left shoulder. On the second and third days there was already noticeable a soft but tender swelling in the muscle and the subcutaneous tissue of the left shoulder; this swelling increased from day to day, and reached its maximum about the end of the week; then it gradually became smaller and firmer. The temperature of both animals was raised on the second and third day, on which days the cows temporarily left off feeding but after this became apparently normal. Both animals exhibited a slight cough, beginning with the eighth to tenth day, and this gradually increased. One animal again left off feeding and ruminating on the twelfth day, "fell in" considerably, and died in the night from the fourteenth to fifteenth day; the other animal, on the twenty-third to twenty-fourth, left off taking food, "fell in" very much, and was very ill; it was killed on the twenty-fifth day.

In both animals, beginning with the fifth day, there appeared on the skin of the udder, less on the teats, red raised papules, which in a day changed into vesicles, surrounded by a rim of injected skin. The contents of the vesicles were a clear lymph; the skin underneath was much indurated, and felt like a nodule; next day the contents of the vesicle had become purulent, *i.e.*, the vesicle had changed into a pustule; in another day the pustule dried into a brownish-black crust, with a sore underneath; this crust became thicker and larger for a couple of days, then became loose, and soon fell off, a dry healing sore remaining underneath. The whole period of the eruption of papules leading to vesicles, then to pustules, and then to brown crusts, which, when falling off, left a dry healing sore behind, occupied from six to eight days. The eruption did not appear in one crop; new papules and vesicles came up on the udder of one cow almost daily between the fifth and eleventh day after inoculation, in the other cow between the sixth and tenth day; the total number of vesicles in the former cow amounted to about 24 on the udder and four on the teats; in the latter they were all on the udder, and amounted to eight in all. The size of the vesicles and pustules differed; some were not more than one-eighth of an inch, others larger, up to one-half to three-quarters of an inch in diameter; they had all a rounded outline, some showed a dark centre. From one of the above cows, on the fifth day, milk was withdrawn from a healthy teat, the outside of the teat and the milker's hand having been previously thoroughly disinfected; from this milk cultivations were made, and it was found that about 32 colonies of the diphtheria bacillus, without any contamination, were obtained from one cubic centimetre of the milk.

With matter taken from the eruption—vesicles and pustules—of the udder, two calves were inoculated into the skin of the groin; here the same eruption made its appearance, red papules rapidly becoming vesicular, then pustular, and then becoming covered with brown-black

* The cows had been kept under observation previous to the experiment for ten days, and were in all respects perfectly normal.

crusts, which two or three days after became loose, and left a dry healing sore behind. More than that, the calves that showed this eruption after inoculation became affected with severe broncho-pneumonia, and with fatty degeneration of the cortex of the kidney. In the two cows above mentioned, on post-mortem examination, both lungs were found highly congested, œdematous, some lobules almost solid with broncho-pneumonia in the upper lobes and the upper portion of the middle or lower lobe respectively; the pleural lymphatics were filled with serum and blood. Hæmorrhages were present in the pericardium and lymph glands, and necrotic patches in the liver. At the seat of inoculation there was in both cases a firm tumour consisting in necrotic diphtheritic change of the muscular and subcutaneous tissue.*

During the past year (1890–1891) six further cows were inoculated in the subcutaneous tissue of the shoulder with recent broth culture of the diphtheria bacillus. In all six cows visceral disease was produced, and at the seat of inoculation a large necrotic tumour; but while in four of these cows there was no eruption in any part of the skin, in the other two there was not only the same kind of eruption on the udder as in the above first two animals, but there was in addition extensive loss of hair in patches and desquamation of the cuticle in these patches. In one of these last two cows, the milk, about the end of the week after inoculation, contained the diphtheria bacillus.

## DISCUSSION.

**Professor Crookshank, M.B.** (London), said that the theory of the origin of human scarlet fever from a disease of the cow had been fully investigated by the Board of Agriculture; but not the least evidence had come to light to support the theory. An outbreak of scarlet fever had been alleged by the officers of the Local Government Board to originate from cows at Hendon suffering from a vesicular disease of the teats communicable from one animal to another by the hand of the milker. The disease, according to Dr. Cameron, was one which produced vesicles on the hands of the milker, but scarlet fever amongst the consumers of the milk. The theory of its producing scarlet fever was based upon the absence, according to Mr. Power, of any of the ordinary sources of infection from a human source. The possibility of infection did exist, for scarlet fever existed at no great distance from the dairy. It was also admitted that the disease was introduced from Derbyshire, and the same disease from the same herd was at the same time conveyed to other dairies; but in no other case was scarlet fever associated with the distribution of the milk. If this line of inquiry had been followed by the officers of the Local Government Board, we should not have had the Hendon case brought forward as an illustration of the preconceived theory of scarlet fever from the cow. With regard to the pathological evidence brought forward by Dr. Klein, it involved the belief in the discovery of the contagium of scarlet fever, and this he (the speaker) could not accept. The organisms described by

* See my paper in the *Proceedings of the Royal Society*, vol. 48.—E.K.

Dr. Klein were in his (Professor Crookshank's) opinion the bovine and human varieties of *Streptococcus pyogenes*, and Continental bacteriologists agreed that this organism was found occasionally in scarlet fever and other diseases, and was not the contagium of those diseases but associated with their complications. He (Professor Crookshank) had suggested that the Hendon cow disease was cow-pox. The suggestion had been received by many with surprise, on the ground that cow-pox was extinct in this country. That it was not extinct was subsequently admitted, for an outbreak in Gloucestershire had been put to the crucial test, with the result of producing typical vaccine vesicles in children, and the lymph stock had been in use for several months. With regard to scarlet fever in man, he said that some species of animals had the privilege of being the natural soil of certain diseases. Swine fever was a disease of the pig; pleuro-pneumonia of cattle; glanders of the horse; and man was the natural soil of scarlet fever and measles, among other diseases. Though the milk of the diseased cows could not, in his opinion, produce scarlet fever, still the researches of the Local Government Board were most important in drawing attention to the contamination of milk which rendered it unwholesome.

**Dr. Ostertag** (Stuttgart) was of opinion that scarlet fever was not communicable from man to animals. Dr. Loeffler had inoculated animals with blood from scarlet fever patients, but had never produced the disease in the cow. With regard to Dr. Klein's experiments, the general opinion in Germany was that Dr. Klein had confounded the disease in question with either cow-pox or foot-and-mouth disease.

**Professor McFadyean** (Edinburgh) said that notwithstanding Dr. Klein's masterly exposition and interpretation of the facts connected with this subject, he remained incredulous that scarlatina was a disease of the cow. He characterised as fallacious the inference that one was justified in saying with reference to any outbreak that no source of human disease could have existed because a careful investigation failed to trace it. Dr. Klein had fallen into error regarding the so-called Edinburgh outbreak, and had been the means of leading others into the same error. He cited that outbreak as an instance of the transmission of a disease of the cow to human beings consuming the milk, but if he would refer to Dr. Cotterill's paper on the outbreak, and to the discussion that followed it before the Edinburgh Medico-Chirurgical Society, he would find that Dr. Cotterill himself did not contend that the infection came from the cow. Indeed, it was clearly brought out that the disease existed in the Edinburgh byre long after Dr. Cotterill supposed that it had ceased, and that milk from cows so diseased was for weeks consumed in the raw state without producing any sore throat or other bad effect.

**Professor Walley** (Edinburgh), whilst complimenting Dr. Klein on the excellence of his paper, differed from him in his conclusions. In reference to scarlet fever it seemed to him that the proof usually brought forward was simply one of exclusion. He pointed out that Dr. Klein's difficulty in tracing cows might be met by an universal system of registration of all cows sold by public auction. One great difficulty he (Professor Walley) had in accepting the statement that scarlet fever in man and eruptive disease in the udder of the cow were identical, was the fact that during an experience of 35 years he had never yet seen a case of disease in the cow that bore any resemblance whatever to scarlet fever. Nor could he understand any specific disease showing different clinical

lesions in two different animals. He further pointed out that too much had been made of the differential characteristics of eruptions, and that such characters could not be relied on unless the case could be watched from beginning to end, and unless extraneous imitation of the eruption could be accomplished. He was pleased to hear Dr. Crookshank refer to the frequent presence of an accidental germ in the products of eruptions, viz., the *Streptococcus pyogenus*, as he was quite sure that such products frequently became so contaminated, as proved by the fact that inoculation with them from different sources produced identical results. He deprecated any idea of wishing to minimise the ill effects of milk from diseased cows, as no man had suffered more in this respect than he had; but he also thought that if the poor cow suffered from all the diseases that had been attempted to be saddled on her, she would have been extinct by this time. Dr. Klein had differentiated three forms of eruptive disease, viz., the "Hendon," the "Wiltshire," and the "Edinburgh," and yet he showed that the same internal lesions existed in the Edinburgh and the Hendon cases. He (Professor Walley) endorsed all that Professor McFadyean had said in reference to the Edinburgh disease, and pointed out that he himself had carried out practical experiments with calves and pigs with negative results. He thought that eruptive disease of the udder of the cow should be scheduled as a contagious disease so far as the consumption of the milk was concerned. In reference to diphtheria, he could certainly not accept Dr. Klein's conclusions without confirmation, as he did not forget the statements made by him some time ago in reference to sheep-pox—which were subsequently shown by Dr. Creighton to be wrong.

**Dr. Henry E. Armstrong** (Newcastle-on-Tyne) said that in investigating the causes of outbreaks of disease in connexion with milk-supply, medical officers of health had in their action to be careful to avoid letting themselves be too easily guided either by pure theory on the one hand, or on the other by the interests of those who were affected by any action arising from putting theory into practice. Among the points relating to the weighing of evidence in inquiries into milk outbreaks which were of practical import, one was that of the denudation of the hair of cows and its value as a sign of scarlet fever. In a scarlet fever outbreak of which he had had experience, where he found that several of the cows presented this sign, he had been able, by the assistance of Mr. Clement Stephenson, F.R.C.V.S., to arrive at the conclusion that this loss of hair was due to the rubbing of the cattle against the posts in the field. In this case also one of the servants at the dairy had sore throat, which,—although at the time he had regarded it as merely Follicular Tonsillitis,—he had since had reason to suspect was in reality scarlatinal. Another point in the investigation of the disease at present known as scarlet fever was that of the nature of the disease itself. Were all the so-called cases of that disease really scarlet fever? After about 20 years' observation of the disease in hospital, he had reason to think that there might be another disease resembling scarlet fever in many of its symptoms, but in reality not the same. This raised the question "What is scarlet fever?" to which the answer was at present somewhat difficult.

**Mr. W. Hunting, F.R.C.V.S.** (London), whilst admiring Dr. Klein's paper as a clever work of art, differed from other speakers in their praise of it. He held that its plausibility was its great danger, as likely to spread error. Dr. Klein denied that the Hendon cow disease was

cow-pox, and of course, if it was, all his theory fell to the ground. Now the disease, whatever it was, existed in four other cow-sheds, and in each was seen by Mr. Villar, V.S. He diagnosed the disease as cow-pox. and although Dr. Klein was an acknowledged authority on bacteriology, he preferred the opinion, on a clinical case in animals, of Mr. Villar. The veterinarian might be content to take a second place in microscopical observation, but it had yet to be shown that he was not a safer guide than any medical man in the practical work of recognising disease in the lower animals. Dr. Klein believed that he had proved that scarlet fever in man had its analogue in cows in the form of an eruptive disease in the udder The veterinarian denied that, and said that no connection had been proved. Milk undoubtedly might carry the infection of scarlet fever, but only as the result of accidental contamination from the human subject. If the two diseases had any connection as cause and effect, they should expect them to prevail synchronously. When scarlet fever raged in a district they ought to find the udders of cows in that district diseased. They found nothing of the sort. Mr. Duguid, when scarlet fever prevailed in many districts of London, examined over 600 dairy-sheds and found no eruptive disease. Mr. Villar and Professor Axe, when an eruptive disease similar to that at Hendon existed in five other dairies, made careful inquiries for scarlet fever in the district to which the milk was distributed. They found none. Again, in other well-recognised diseases of animals communicable to man, they found the symptoms exhibited by both patients nearly alike and communicable to and from either. In this case Dr. Klein would have them believe that such a disease as scarlet fever in man—a distinct and definite general affection—was represented in cows by an eruption on the skin of the udder. Veterinarians recognised no such disease as scarlet fever in animals. Professor McFadyean had tried by every possible plan to produce it. He had taken scarlet fever matter, and introduced it into the system of cows and calves by ingestion, by inhalation, by inoculation, but with no definite result. The only logical inference to be deduced from the facts and experiments which had been made by scientists of all kinds was that Dr. Klein had made a mistake. The clinical observations of capable observers were entirely opposed to him, and even his bacteriological work was contradicted by an equally trustworthy investigator, Dr. Crookshank. He submitted that an understanding of the relation of the diseases of man to those of animals required clinical, medical, and veterinary observers to act together, and that until such co-operation could be obtained, they would have unnecessary scares sprung upon the public, and erroneous deductions forced upon them by bacteriologists.

**Dr. Geo. Turner** (Broxbourne), Medical Officer of Health for East Hertfordshire, said it was impossible to state with certainty that scarlet fever or diphtheria was absent from a neighbourhood. They could only assert that, having taken all possible means to discover it, no case had been brought to light. The question was much obscured by trade interests as well as by medical interests. Symptoms differed much and were not similar in different animals. A transformation had been going on for nearly one hundred years. Small-pox, an infectious disease accompanied by a general eruption in man, was in the cow no longer infectious, was not accompanied by a general eruption, and when returned to its original habitat it did not recover its former characters.

**Dr. W. J. Collins** (London) said that he had listened to the discussion with great interest, more especially as bearing upon the questions

under review by the Royal Commission on Vaccination, of which he was a member. He had listened with amazement to the rapid multiplication of the varieties of contagious diseases derivable from the nipples of the cow under the differentiating capacities of Dr. Klein. The question occurred to him whether, in less discriminating days, some or all of these might not have been put to the use to which Jenner put it. The question was a serious one. Dr. Klein's researches had been severely criticised; if not re-established, the public as well as the professional conscience would be disturbed. If the warmth of the discussion were to be deprecated, at any rate the conflict of the two State Departments had stimulated bacteriological research. Dr. Klein had not mentioned any essential or cardinal criterion for the differentiation of true cox-pox (so-called cow-small-pox), from the increasing variety of other teat eruptions. Was there any bacteriological or other test of true cow-pox, apart from its cultivatibility so as to give a "vaccine" vesicle on a child's arm? The previous speaker stated that an analogy for a teat eruption, giving a human general disease and *vice versâ* was provided in the case of human small-pox causing cow-pox. Did Dr. Klein agree with that? He (Dr. Collins) understood that Dr. Klein's attempts to thus produce a cow-pox signally failed. Did Dr. Klein accept the analogy as supporting his views.?

**Dr. Hime** (Bradford) observed that the subject under discussion was one of the greatest public importance. He had devoted himself for a considerable time to investigating the relationship between scarlet fever and milk in Bradford, a town of 230,000 inhabitants, where scarlet fever was endemic and where a few years ago it was severely epidemic, some fifty to sixty cases being notified weekly under compulsory powers. The Corporation also had power to compel milkmen to give a list of their customers. Usually in inquiries of this kind, it was reported that a certain number of sick persons used the infected milk (or did not), but they seldom had particulars as to all the parties using the milk. He had therefore much useful and necessary information at his disposal as medical officer of health. His attention was called on one occasion to a case of scarlet fever, not reported, which had been discovered in the house of a dairyman. This child had been ill several weeks; the father lived day and night in frequent communication with it. The vessels were washed in the house, and were often washed by this man. The child was sent to hospital, a list of customers was procured, and on examination was found to contain no person reported to have had scarlet fever. Each house was subsequently visited, and no case of scarlet fever could be traced to have existed. In a word, after investigating a large number of similar cases, he had found cases of scarlet fever among milk consumers, when none existed in the dairy; and on the other hand cases of scarlet fever were observed in dairies though none occurred among the consumers. These observations held good for over 4,000 consumers. As a fact, there was a smaller proportion of cases among these 4,000, than among an equal number of inhabitants throughout the borough generally. As an illustration of the unfair and injurious manner in which conclusions were sometimes jumped at, he mentioned one where the milk had been blamed for introducing scarlet fever, but it had not been ascertained that the servant of the house was sister to a nurse in the Fever Hospital, and that these two frequently exchanged visits. In another case, where the milk was similarly condemned, it had escaped notice that a boy was engaged to clean boots who performed the same duties in several other

houses where scarlatina existed. In these cases he utterly refused to admit the probability of the milk being in fault as the source of the infection. In all those cases the cows had been examined. Some had loss of hair, cows and men were equally liable to that; some had sores of various kinds, cows were not exempt from that either. But the fact was that in no case had scarlet fever spread from the dairies, even where the milkman had slept with the infected person. This was not a question of theory: it was a fact, and that state of things prevailed during no less than six months and the cases were watched for that period. Such observations, on such a scale, involving some 4,000 persons, and carried on for such a lengthened time, proved beyond all doubt that the Bradford milk when exposed to every condition which one would adopt who wished to infect it with scarlet fever, had utterly failed to produce that disease; and that in that town the disease was not spread by milk. Who could doubt that there was no specific peculiarity about the population of Bradford, or that the same facts existed among other populations in the country, if the circumstances were only investigated in the same way, and not as was too manifestly often the case, examined with a view to prove that the milk was the source of the disease?

**Dr. A. Bostock Hill** (Medical Officer of Health for Birmingham) said he had had experience of an epidemic of scarlet fever in the spring of the present year occurring in the borough of Sutton Coldfield, which was associated with a particular milk supply, and in which no human infection could be traced. On the 5th of March one or more cases occurred, followed by others daily for about three weeks. All those cases occurred in houses of from 50*l.* to 150*l.* per year in rent, and were supplied from one dairy of about eighteen cows. The milk was distributed by three channels. A small quantity went directly from the dairy. The great bulk of it was, however, distributed by one milkman, while another took a small quantity to supplement his own supply. The Notification Act was in force in the borough, and consequently all the cases could be investigated. There was not the least doubt that the infection was conveyed by the milk. No trace of scarlet fever or of any other illness could be discovered in the dairyman's or the cowman's houses. Attention was turned to the cows, when it was found that one cow recently purchased, and newly calved, was suffering from hairless patches on the quarters, while there was also an ulcer on one of the teats. One other cow in the shed appeared to suffer somewhat at a later stage. The matter was referred to the Health Committee, and the dairyman at their request consented to withhold his milk from sale for a month. Directly after this took place the epidemic ceased. An interesting point in connection with the epidemic was its fatality, the mortality being no less than 12·5 per cent. of the cases, while the age of the patients was also deserving of notice, as, out of 40, no less than six were upwards of 20 years of age, the average age of each patient being 11·2 years. In the previous two years, out of about 100 cases reported, there had not been a single death, and that appeared to point to some new infective material having been introduced.

**Professor Brown, C.B.** (London), said that the speakers who had preceded him had not only taken up all the points which he had noted upon Dr. Klein's paper, but had gone considerably further than he should have done had he spoken at the commencement of the debate. The advantage to him was that he had the benefit of finding that a considerable number

of those speakers had adopted very much the view which he should have taken had he dealt with those points in the first instance. After deprecating Dr. Klein's remarks upon the existence of "rivalry" between the Local Government Board and the Board of Agriculture, and dealing at some length with the relations of the two Departments in reference to outbreaks of scarlet fever, Professor Brown pointed to the possibility of the milk at the time of the Hendon outbreak being contaminated from human sources, as shown by the evidence of the local doctor to the effect that children were to be seen running about the streets whilst recovering from the disease (peeling), and to the fact that some of the dairymen lived at the dairy and were constantly going backwards and forwards to the town.

**Professor Chauveau** (Paris) stated that he had not himself made personal experiments lately in regard to this subject, but he gave details of the results of experiments by his colleagues. Cow-pox was prevalent in France as it was in England, and it was very widely distributed in Germany. In France, too, horse-pox was even more prevalent than cow-pox. Some years ago he had personally made experiments with the object of transmitting the small-pox virus to bovine animals, but he had met with no success whatever. Since then he had been more successful, and as the result of a very considerable number of experiments he had found himself able, in every case attempted, to communicate the small-pox virus to bovine animals. But he had never, in any attempt, succeeded in transforming this cow-pox virus into vaccine.

**The President** said that the crowded state of the room throughout the day, was sufficient evidence of the great interest excited by the discussion on Dr. Klein's paper, and in view of the importance of the subject he had not set any limits to the debate. He thought they would all agree that the three hours which had been devoted to the question had been in no wise wasted, but had been most profitably spent. At the same time, he must remind them that there was another aspect of the milk question still to be considered by the Section, in connection with the paper prepared by Dr. Ostertag, of Stuttgart. They would be aware that, according to the original programme, their Section was to unite next day with Section II., to discuss the question of Tuberculosis in all its bearings. Papers had already been prepared on behalf of Section III., by Dr. Bang, of Copenhagen, and Professors McFadyean and Woodhead, on "The Alleged Danger of Consuming the Apparently Healthy Meat and Milk of Tuberculous Animals." * The Section would undoubtedly have wished to be present whilst these papers were being read and discussed in the meeting-place of Section II. He thought, however, that under the circumstances the consideration of the question of Tuberculosis might very well be left in the hands of Section II., augmented by such members of their own Section as preferred to be present. After conference with his colleagues on the executive he had, therefore, decided to ask Section III. to meet in its own hall on the following day to discuss the two questions of the Regulation of the Milk Trade and the Inspection of Meat. If time remained, after these subjects had been disposed of, the Section would unite with Section II. for the remainder of the day, to consider the question of Tuberculosis.

---

* For the text of these papers, and a report of the discussion thereon, see Volume II., pp. 193 *et seq.*

## Thursday, 13th August 1891.

The PRESIDENT, Sir NIGEL KINGSCOTE, K.C.B., in the Chair.

VICE-PRESIDENTS of the day:
Professor E. PERRONCITO, Turin.
GEORGE FLEMING, C.B., LL.D., late Principal Veterinary Surgeon of the Army, London.

## Die Regelung der Milchversorgung mit Hinsicht auf übertragbare Krankheiten.

VON
Professor Dr. OSTERTAG, Stuttgart.

Die Regelung der Milchversorgung mit Hinsicht auf übertragbare Krankheiten lässt in den meisten Ländern noch viel zu wünschen übrig. Die Mehrzahl der Culturstaaten glaubt in dieser Frage ihre Pflicht erfüllt zu haben, wenn sie den Consumenten vor financieller Schädigung durch regelmässige Kontrolle der Marktmilch auf ihren Fettgehalt zu schützen suchen. Der ungleich wichtigeren sanitären Seite des Milchverkehrs wird gemeinhin geringere Beachtung geschenkt. Nicht als ob diese Seite in den auf den Milchhandel bezüglichen Verordnungen gänzlich unberücksichtigt geblieben wäre. Nein, man findet regelmässig in den Milchhandelsverordnungen einen Paragraphen, welcher das Inverkehrbringen von abnormer oder von kranken Kühen stammender Milch unter Strafandrohung verbietet. Allein dieses Verbot ist nur wenig wirksam, weil es im Gegensatz zu der auf Milchverfälschung gerichteten Kontrolle an Ausführungsbestimmungen fehlt, welche eine regelmässige sachverständige Kontrolle des milchwirtschaftlichen Betriebes vorschreiben. An der auf den Markt gebrachten Milch ist es aber erfahrungsgemäss ein Ding der Unmöglichkeit, gerade die gesundheitsschädliche Beschaffenheit, sei es in Folge irrationeller Fütterung oder von Krankheiten der Milchtiere, zu erkennen. Lediglich die sogennanten Milchfehler lassen sich an der Marktmilch feststellen. Diese bedingen aber just keine Gefahr für die Gesundheit der Menschen.

An einem Beispiel möge der heutige Stand der Regelung der Milchversorgung im Königreiche Preussen erläutert werden. Die deutsche Reichsregierung war im Jahre 1882 der Frage nähergetreten, ob der Milchhandel nicht auf Grund des Nahrungsmittelgesetzes vom 14. Mai 1879 einheitlich zu regeln sei. Von einer einheitlichen Regelung wurde aber abgesehen, weil eine für das ganze deutsche Reich bindende Festzetzung des niedersten Grenzwertes für den Fettgehalt der Milch wegen der wirtschaftlichen Verschiedenheiten in den einzelnen Distrikten nicht für angängig erachtet wurde. In Folge dessen gaben die

preussischen Ministerien des Innern, des Kultus und für Landwirtschaft den Bezirksregierungen anheim, die beregte Frage für ihre Bezirke nach Massgabe besonders bezeichneter Gesichtspunkte zu ordnen. Hierauf wurden in einer grossen Anzahl von Städten der Monarchie, durchaus nicht in allen, Polizeiverordnungen betreffend den Milchverkehr erlassen. Als Muster einer solchen Verordnung führt Kirchner (Handbuch der Milchwirtschaft) diejenige für die Stadt Celle an. Dieselbe besteht aus fünf Paragraphen. § 3 behandelt die hygienische Seite des Milchverkehrs und lautet folgendermassen :—" Vom Handelsverkehr im gesundheitspolizeilichen Interesse ausgeschlossen ist die ganze oder abgerahmte Milch, welche von kranken, insbesondere mit irgend welcher Seuche behafteten Tieren oder von Kühen innerhalb der ersten Woche nach dem Kalben abstammt, ferner jede bittere, schleimige abnorm gefärbte oder Ekel erregende Milch." Ein Reglement zu dieser Polizeiverordnung befasst sich nur mit der Ausübung der Untersuchung der Marktmilch auf den Fettgehalt, gedenkt aber mit keinen Worte der Haltung der Milchtiere, der sachverständigen Ueberwachung des Betriebes und der Behandlung der Milch nach dem Melken. Da, wie schon erwähnt, gesundheitsschädliche Beschaffenheit und im Besonderen die Herkunft von kranken Tieren an der zu Markt gebrachten Milch nicht mehr nachgewiesen werden kann, so steht der angeführte Paragraph der Celler und aller ähnlichen Verordnungen lediglich auf dem Papier. Verfehlungen gegen denselben gelangen nur durch Denunciationen zur Kenntniss der Behörden. Die Zahl dieser Fälle steht aber, wie jeder mit den Verhältnissen namentlich kleinerer Milchwirtschaften Vertraute zur Genüge weiss, in keinem Verhältniss zu der Anzahl der thatsächlich vorkommenden Vergehen gegen obige Vorschrift. Aehnlich wie die angezogene Verordnung für die Stadt Celle lautet nach Kirchner das schweizerische Reglement über den Milchverkehr. In hocherfreulichem Gegensatz zu diesen und ähnlichen Verordnungen steht das Reglement, welches in Königreich Italien unter dem 3. August 1890 betreffend die sanitäre Überwachung der Nahrungsmittel, Getränke u.s.w. als Grundlage für die Ortsgesundheits-Reglements der Einzelbezirke erlassen worden ist. Die auf Milch, Butter und Surrogate, Käse und Milchspeisen bezüglichen Vorschriften müssen im Allgemeinen als mustergiltige bezeichnet werden.

Der Staat hat die unabweisbare Verpflichtung, dafür Sorge zu tragen, dass nur *gute* Milch in den freien Verkehr gelange, weil der Consument nicht imstande ist, sich vor den mannigfachen Gefahren zu schützen, welche mit dem Genuss von Milch, dem täglichen Nahrungsmittel, verbunden sein können. Trotz weisser Farbe und süssem Geschmack, dieser Kriterien der Güte für den Laien, kann Milch die gesundheitsschädlichsten Eigenschaften besitzen. Solche schädliche Milch kann nur dann aus dem Verkehre wirksam verbannt werden, wenn nicht bloss der Milchhandel, sondern auch die Milchgewinnung einer sachverständigen Überwachung unterliegen.

Im freien Verkehre darf nur *gute*, d. h. mit der grössten Sauberkeit von gesunden Tieren unter rationellen Fütterungsverhältnissen

gewonnene Milch geduldet werden, welche normale physikalische Eigenschaften und eine gewisse Haltbarkeit besitzt. Vom Verkehre dagegen muss ausgeschlossen werden :—

(1.) Milch, welche, ohne gesundheitsschädlich zu sein, Abweichungen in Farbe, Geschmack oder Consistenz zeigt (verdorbene Milch).

(2.) Alle Milch, welche *gesundheitsschädlich* ist oder bezüglich welcher der begründete Verdacht besteht, dass sie gesundheitsschädlich sei.

Zu der Gruppe (1.) gehört die Colostralmilch, die blutige, blaue, rote, gelbe Milch, ferner die schleimige, fadenziehende, bittere, salzige, sowie abnorm riechende und die mit Schmutz oder anderen Stoffen verunreinigte Milch. Diese ganze Gruppe kann hier aber unerörtert bleiben, da trotz gegenteiliger Angaben in der Litteratur angenommen werden muss, dass die zu derselben zählenden Milchabnormitäten keine Gesundheitschädlichkeit für den Menschen besitzen.

Ueber gesundheitsschädliche Milch ist in jüngster Zeit eine Anzahl, zum Teil recht gründlicher Arbeiten erschienen, welche ein beredtes Zeugniss von dem gesteigerten Interesse an dieser Frage ablegen. Von diesen Arbeiten nenne ich besonders Schmidt-Mülheim "Über die Aufgaben der Veterinärmedicin auf dem Gebiete der Milchhygiene;" (Archiv für animalische Nahrungsmittelkunde, Band I.); Marsæ, "Die gesundheitspolizeiliche Ueberwachung des Verkehrs mit Milch;" (Deutsche Vierteljahresschrift für öffentliche Gesundheitspflege, Band XXII.); Petersen, "Über die Verbreitung ansteckender Krankheiten durch Milchgenus;" (Tiermedicinische Vorträge, II. Band); Sonnenberger, "Die Enstehung und Verbreitung von Krankheiten durch gesundheitsschädliche Milch;" (Deutsche, Medicinische Wochenschrift 1890); Würzburg, "Über Infectionen durch Milch;" (Therapeutische Monatshefte, 1891); und Fröhner, "Über die Bedeutung der Milchmittel;" (Monatshefte für praktische Tierheilkunde, Band II.). Unter Hinweis auf diese Arbeiten, welche sorgfältige Literaturstudien enthalten, kann ich es mir hier versagen, bei der Erörterung der Einzelfragen der Gesundheitsschädlichkeit der Milch genauere Literaturangaben zu machen.

Gesundheitsschädliche Beschaffenheit kann die Milch annehmen bei irrationeller Haltung und zwar: (1.) bei Benutzung von Heu, beziehungsweise von Weiden, welche reichliche Mengen von *Giftpflanzen* aufweisen; (2.) bei der Verfütterung *gewisser gewerblicher Rückstände.* Nach den Angaben der Literatur wurden ruhrartige Durchfälle beobachtet nach dem Genuss der Milch einer Ziege, welche *Euphorbiumarten* gefressen hatte. Die Milch von Kühen, welche *faulige Rübenblätter* erhalten hatten, erzeugte Brechdurchfall bei Kindern. Nach dem Genusse von *Schlempemilch* sah man Wundsein und Nässen in den Hautfalten der Säuglinge. Indessen wird von dieser Milch unter Umständen eine weit schädlichere Wirkung angenommen, weil die tierärztliche Erfahrung lehrt, dass Kälber, welche mit Schlempemilch gefüttert werden, daran zu Grunde gehen. Aehnliche Beobachtungen bei Kälbern liegen für die Verfütterung der *Rübenpresslinge*

und der *Melasse* vor. Schmidt-Mühlheim giebt unter Betonung des hohen Kaliumgehaltes der Melasse (10 %) an, dass schon Mengen von 2–3 Pfund täglich genügten, die Milch so ungesund zu machen, dass auch nicht ein einziges Kalb mehr aufgezogen werden konnte. Derart gewonnene Milch darf auch nicht im Verkehr als Nahrungsmittel für Menschen geduldet werden. Aber nicht nur die Verfütterung unverhältnissmässiger Mengen von Schlempe und Melasse geben der Milch eine giftige Beschaffenheit, dieselbe wird auch beobachtet bei der Verwendung der *Rückstände bei der Oelfabrikation* als Futter für Milchkühe. Nach Erdnusskuchen ist, wie Schmidt-Mühlheim angiebt, zuweilen eine abführende Milch bei Kindern eingetreten; für noch viel gefährlicher wegen ihres Gehaltes an Ackersenf hält aber derselbe Autor die Raps- und Rübenkuchen. Wenigstens seien nach der Verfütterung der letztangeführten Kuchen bei den Kühen heftige Entzündungen des Verdauungs- und Harnapparates festgestellt worden, und nach dem Genusse der Milch seien schon Kälber an tödlichen Durchfällen erkrankt. Bollinger berichtet über die Schädlichkeit der Milch nach der Verfütterung von Ricinuskuchen.

Die Vorbeuge gegen die angeführten Milchschädlichkeiten ergiebt sich von selbst. Es muss für Beseitigung der Giftpflanzen auf den Wiesen gesorgt, die Verfütterung absolut nachteiliger technischer Rückstände (zum Beispiel Melasse, Raps- und Rüben-, ferner Ricinuskuchen) sowie verdorbener Rückstände an Milchkühe ganz verboten, die Verfütterung der übrigen aber nur in richtigem Verhältniss mit Heufütterung erlaubt werden. Denn ganz auf die Verwertung technischer Rückstände Verzicht zu leisten, erscheint aus wirstchaftlichen Gründen nicht angängig.

Derselben Beurteilung, wie die Milch von Kühen, welche mit giftig wirkenden Futterstoffen ernährt werden, unterliegt die Milch mit *scharf wirkenden Medicamenten* behandelter Tiere. Für eine ganze Reihe von Arzneistoffen ist der Übergang in die Milch festgestellt. Kampfer, Terpentinöl und Kamillen machen sich rasch in der Milch bemerkbar. Der Uebergang von Arsen in die Milch, ebenso wie von Blei, und zwar zum Teil *in giftigen Mengen*, ist durch mehrfache Beobachtungen sichergestellt. Erwiesen ist ferner der Uebergang von organischen Giften, ausserdem von Jod, Eisen, Zink, Wismuth, Antimon, von Brechweinstein, Kupfer, und Quecksilber. Nach der Verabreichung von Aloë erhielt die Milch nicht nur einen bitteren Geschmack, sondern wirkte auch bei Kindern schädlich. Ausserdem wurden Erkrankungen nach dem Genus kupfer- und quecksilberhaltiger Milch beobachtet. Über die Milch medicamentös behandelter oder vergifteter Tiere sagt Fröhner, welcher in Verbindung mit Knudsen die Unschädlichkeit des Fleisches vergifteter Tiere festgestellt hat, im Allgemeinen, dass *die Milch im Gegensatz zum Fleische nach Einverleibung von Arzneimitteln unter Umständen gesundheitsschädlich werden könne.*

Diese Thatsachen rechtfertigen den Ausschluss aller Milch von Tieren, welche mit toxisch wirkenden Medicamenten behandelt werden.

Eine dritte Ursache für gesundheitsschädliche Beschaffenheit der Milch, geben *Erkrankungen der Milchtiere* ab. Hierbei kommen hauptsächlich in Betracht septische Allgemeinerkrankungen, die Aphthenseuche und Tuberkulose. Kurze Erwähnung verdienen die Wut, die Pocken und der Milzbrand. Die Wut und die Pocken der Kühe gehören zu den grossen Seltenheiten. Eine Übertragung dieser Krankheiten durch Milchgenuss ist mit Sicherheit nicht festgestellt. Bei dem Milzbrand kann die Milch virulente Eigenschaften besitzen. Ueber Milzbrandinfection beim Menschen, welche auf diese Weise zu Stande gekommen wäre, ist aber nichts bekannt. Indessen sei daran erinnert, dass sehr haüfig auch das Fleisch milzbrandiger Tiere (trotz seines massenhaften Bakteriengehaltes) ohne jeglichen Nachteil von Menschen verzehrt worden ist. Im Uebrigen spielt der Milzbrand in unserer Frage eine ganz untergeordnete Rolle, weil das Versiegen der Milch zu den ersten Symptomen dieser Krankheit gehört. Von der Lungenseuche des Rindviehs ist behauptet worden, dass dieselbe durch Milchgenuss auf den Menschen, speciell auf Kinder übertragen werden könne. Diese Behauptung wird jetzt allgemein als nicht zutreffend angesehen, und wohl mit gutem Rechte. Denn sonst müssten in den Lungenseuchedistrikten, in welchen—vor amtlicher Feststellung der Seuche—jahraus jahrein gross Mengen von Milch lungenseuche kranker Tiere getrunken werden, diese Erkrankungen häufiger zur Beobachtung kommen. Beim Starrkrampf der Kühe ist anzunehmen, dass die toxischen Stoffe des Tetanusbacillus auch in die Milch übergehen. Nach den Versuchen Sormani's aber dürfte gleichwohl eine schädliche Wirkung solcher Milch nicht eintreten, da per os eine zehntausendmal grössere Menge von tetanogenem Virus eingeführt werden kann, als diejenige, welche bei der Einführung unter die Haut den Tod bedingt.

Trotzdem nun bei Wut, den Pocken, dem Milzbrand, der Lungenseuche und dem Starrkrampf eine gesundheitsschädliche Beschaffenheit noch nict beobachtet wurde und die Möglichkeit einer solchen zum Teil als ausgeschlossen, zum anderen Teil als eine sehr geringe betrachtet werden muss, ist der Verkauf der Milch bei diesen Krankheiten durchweg zu verbieten, weil dieselbe in Folge ihrer Abstammung von schwerkranken Tieren als ein ekelerregendes Nahrungsmittel anzusehen ist. Der wirtschaftliche Verlust, welchen der Ausschluss dieser Milch bedeutet, ist dazu ein ganz untergeordneter, weil die beregten Infectionskrankheiten—von dem Milzbrande und der Lungenseuche abgesehen—nur selten vorkommen und die Milchproduction dieser Tiere stets verringert, wenn nicht ganz aufgehoben ist.

Als hygienisch wichtiger zu betrachten sind *septische Erkrankungen der Kühe,* über deren Wesen noch ein gewisses Dunkel herrscht. Die bekannten Fleischvergiftungen, welche leider nicht allzuselten zur Beobachtung kommen, liefern den Beweis, dass bei den Rindern septische Erkrankungen vorkommen, welch durch Genuss des von diesen Tieren stammenden Fleisches auf den Menschen übertragbar sind. Ich erinnere nur an die in letzter Zeit beobachten Vergiftungen in Frankenhausen, in Cotta, Löbtau, in Kirchlinde und Frohlinde.

Alle diese Fleischvergiftungen, welche zur Erkrankung selbst hunderter von Personen führten, erfolgten nach dem Genusse des Fleisches von schwer erkrankten ***Kühen.*** In einem Falle (Cotta) war der Ausgangspunkt der Allgemeinerkrankung eine Entzündung des Euters. Obwohl nun keine ausdrücklich vermerkte Beobachtung die Annahme erhärtet, dass auch die Milch ebenso wie das Fleisch der fraglichen Tiere gesundheitsschädlich gewirkt habe, so ist die Wahrscheinlichkeit hierfür doch sehr gross, namentlich bei denjenigen Fleitschvergiftungen, bei welchen nicht nur eine schädliche Wirkung durch Übertragung specifischer Bakterien, sondern auch durch Aufnahme chemischer Stoffwechselprodukte festgestellt worden ist. Denn letztere werden sicherlich durch das Euter ausgeschieden. Genauere Untersuchungen liegen nur über die Fleischvergiftungen in Frankenhausen (Gärtner) und in Cotta (Johne) vor. In beiden Fällen wurde als Ursache der Vergiftung der Bacillus enteritidis Gärtner gefunden. Im Allgemeinen scheinen die zu Fleischvergiftungen führenden Erkrankungen der Rinder unter den verschiedensten Krankheitsbildern auftreten zu können. Sie dürften aber nach den vorliegenden Mitteilungen, soweit dieselben auf Zuverlässigkeit Anspruch erheben können, das Gemeinsame des hohen consumierenden, mit Prostration der Kräfte einhergehenden Fiebers bezitzen. Weil somit einzelne Krankheitsformen, welche hier in Betracht zu ziehen sind, nicht besonders namhaft gemacht werden können, muss das ***allgemeine Verbot des Verkaufs der Milch fieberhaft erkrankter Tiere*** wegen Verdachts der Gesundheitsschädlichkeit angeordnet werden. Auch hier ist der nationalökonomische Schaden nicht erheblich, weil fieberhaft erkrankte Tiere erfahrungsgemäss nur wenig Milch producieren. Das Verkaufsverbot ist durch die ***Anzeigepflicht*** bei vorkommenden Krankheiten unter dem Milchvieh zu unterstützen, wie dieses in dem italienischen Milch - Reglement vorgeschrieben ist. Denn selbst die wenige, von kranken Tieren producirte Milch wird in der Regel von den Milchwirten aus freien Stücken nicht weggegossen.

Die ***Aphthenseuche*** spielt wegen der Häufigkeit ihres Auftretens und der grossen Anzahl von Tieren, welche bei jeder Epizootie befallen werden (bis zu 10 % des ganzen Rindviehbestandes eines Landes und darüber), eine wichtige Rolle in der Milchhygiene. Durch Versuche und Beobachtungen ist der sichere Beweis erbracht, dass durch den Genuss roher Milch eine der Aphthenseuche entsprechende Krankheit übertragen werden kann. Namentlich haftet die Krankheit bei Kindern; indessen können auch Erwachsene inficirt werden. Bei Kindern werden sogar Todesfälle auf den Genuss der Milch von maul-und klauenseuchekranken Tieren zurückgeführt, und es klingt bei der zeitweise enormen Verbreitung der Krankheit ganz glaubhaft, wenn Wyss angiebt, er habe ***förmliche Epidemieen*** der Aphthenkrankheit unter Kindern beobachtet. Indessen ist erwiesenermassen nur ***rohe*** Milch schädlich. Einfaches Kochen zerstört das specifische Gift. Wegen dieser geringen Resistenz des Aphthenseuchevirus ist eine völlige Ausschliessung der Milch aphthenseuchekranker Tiere, wie sie zum Beispiel Sonnenberger will und das Berliner Polizei-Präsidium,

ganz im Gegensatz zu der Bestimmung des Deutschen Reichsviehseuchengesetzes anordnet, nicht notwendig. Dass die Milch von aphthenseuchekranken Tieren im gekochten Zustande völlig unschädlich ist, zeigt nicht nur die landläufige Erfahrung, sondern auch eine genaue Untersuchung, welche Cnyrim anlässlich der im Jahre 1884 in der Milchkuranstalt zu Frankfurt a. Main ausgebrochenen Maul- und Klauenseuche angestellt hat. Er stellte fest, dass bei den Kindern, welche die Milch aus der verseuchten Anstalt in gekochtem Zustande weitergenossen, weder eine specifische Erkrankung noch irgend ein anderer Nachteil eintrat. Dasselbe kann ich selbst auf Grund einer im Jahre 1889 in einer Berliner Milchkuranstalt beobachten Seuche vollauf bestätigen.

Mithin braucht bei der Aphthenseuche der Rindviehs nur der Verkauf der *rohen* Milch verboten zu werden. Das Inverkehrbringen der gekochen oder sterilisirten Milch dagegen ist zu gestatten.

*Tuberkulose.*—Diese Krankheit muss wegen ihrer Ausbreitung unter den Kulturrassen als die grösste Calamität unserer Rindviehzucht bezeichnet werden. Nach Ausweis der Schlachthausberichte beträgt die Zahl der tuberculösen Rinder insgesammt 5-10 %. Etliche Schlachthäuser registriren weit höhere Zahlen, zum Beispiel verzeichnet Leipzig (1885) 15 % (in einem Monat 22 %); Stolp 20·7 %; Bromberg 26·2 %. Hierbei ist noch zu berücksichtigen, dass die Procentziffer der Erkrankung der Kühe eine beträchtlich höhere ist, als bei den übrigen Rindergattungen. So waren zum Beispiel in dem zuletzt genannten Schlachthause Bromberg 36·02 %, also mehr als ein Drittel aller Kühe mit Tuberkulose behaftet. Nach meinen eigenen, während 6 Jahren auf dem Schlachthofe zu Berlin gesammelten Erfahrungen ist die durchnittliche Tuberkuloseziffer eine höhere, als allgemein angegeben wird, wenn man auch diejenigen Fälle in Rechnung zieht, welche nur an dieser oder jener Eingangspfort eine tuberkulös erkrankte Lymphdrüse zeigen. Diejenigen Schlächter, welche ausschliesslich ältere, abgemolkene Kühe schlachten, sind es gewöhnt, dass ihnen sämmtliche oder jedenfalls 53·7 % aller Lungen wegen tuberculöser Veränderungen dieser selbst oder der Bronchial- beziehungsweise Mediastinaldrüsen confiscirt werden. Diese Zahlen gewinnen ein erhöhtes Interesse durch die Feststellung (Hirschberger), dass bei 55 % der darauf untersuchten Kühe die Milch sich virulent zeigte. Wenn nun auch Bang nur bei 17·7 % tuberkulöser Kühe Virulenz der Milch nachweisen konnte, und wenn wir weiterhin bedenken, dass Virulenz bei intraperitonealer Meerschweinchenimpfung noch nicht gleichbedeutend ist mit Infectiosität dem Milch bei dem Genusse durch Menschen, so muss immerhin bei der grossen Verbreitung der Tuberkulose unter den Rindern diese Krankheit als eine Gefahr für die Gesundheit des Menschen bezeichnet werden. Eine ohne jeden Zweifel aber *ganz bedeutende Gefahr* schliesst die Milch derjenigen Kühe ein, welche *mit tuberkulöser Erkrankung des Euters* behaftet sind. Kein tuberkulöses Produkt beim Rinde weist so massenhaft Bakterien auf, wie das Sekret eutertuberkulöser Tiere, und dem entsprechend zeigt auch die Milch solcher Kühe, wie Bang gezeigt

hat, auch eine ganz bedeutende Virulenz. Bei der Eutertuberkulose sind folgende wichtige Punkte zu bedenken:—(1.) befällt sie nicht das ganz Euter, sondern lässt in der Regel ein oder mehrere anscheinend gesund Viertel übrig; (2.) ist die Milch dieser anscheinend intacten Viertel nach den Festellungen Bang's ebenfalls virulent; (3.) kommt die Eutertuberkulose bei einem immerhin noch recht beträchtlichen Procentsatze tuberkulöser Tiere vor. Bang konnte zum Beispiel im Verlaufe mehrerer Monate nicht weniger als 27 Fälle in den Milchwirtschaften Kopenhagens feststellen, und nach dem amtlichen Berichte für das Königreich Sachsen im Jahre 1888 und 1889 zeigten 4 % besugsweise 3·6 % der tuberkulösen Tiere Eutertuberkulose. Diese Zahlen stimmen ungefähr mit meinen Erfahrungen von dem Schlachthofe zu Berlin überein. Der Umstand, dass die tuberkulöse Euterentzündung sich allmählich und ohne Schmerzen entwickelt, und dass noch anscheinend normal secernirende Viertel übrigbleiben, macht die tuberkulöse Euterentzündung zu einer *gemeingefährlichen* Krankheit. Denn jedem, welcher die gewöhnlichen milchwirtschaftlichen Verhältnisse kennen gelernt hat, dürfte es bekannt sein, dass die Milch derartiger Kühe nicht in toto weggegossen wird, sondern solange in den Verkehr gelangt, als sie anscheinend normale Beschaffenheit besitzt. Hiefür spricht auch ganz unzweideutig die von mir auf dem Berliner Schlachthofe nicht selten gemachte Beobachtung, dass Kühe, welche mit Tuberkulose eines oder zweier Euterviertel behaftet waren, noch zirmlich stark laktirende Restviertel bessassen. Die Eutertuberkulose ist angesichts der Thatsache, dass noch sehr viel Milch ungekocht genossen wird, ein mehr als hinreichender Grund, *regelmässige tierärztliche Kontrolle der Milchkühe* zu verlangen. Die Milch eutertuberkulöser Tiere ist ein eminent gefährliches Gift, und das Inverkehrbringen eines solchen Giftes, welchem bei der Eigenart des Milchhandels zahlreiche Menschenleben zum Opfer fallen können, muss wie dasjenige der medicamentösen Gifte staatlicherseits ohne Rücksicht aufswirtschaftliche Bedenken verhindert werden. Das staatliche Einschreiten wäre ohne Zweifel schon längst geschehen, wenn tuberkulöse Mich eine sichbare, sogleich nach dem Genusse auftretende schädliche Wirkung entfalten würde wie die übrigen Gifte. Aber gerade mit Rücksicht auf die heimtückiche schleichende Art der dauernden Gesundheitsgefährdung durch tuberkulöse Infection sollte die Kontrolle der Milchtiere eine um so verschäftere sein.

Wenn es nun auch nicht dem geringsten Zweifel unterliegen kann, dass die Milch eutertuberkulöser Kühe *unbedingt* vom Consume ausgeschlossen werden muss, so liegen die Verhältnisse bezüglich derjenigen Tiere, welche der Tuberkulose innerer Organe verdächtig sind, ohne dass sie evidente Erscheinungen, namentlich auch des Euters zeigen, nicht so einfach. Die Frage der Diagnostik der Tuberkulose beim lebenden Tiere ist durch die Koch'sche Entdeckung in ein neues Stadium getreten. Soweit die vorliegenden, bereits recht umfangreichen Versuche eine Urteil gestatten, scheint die Annahme wohl bergründet zu sein, dass es mittels der Impfung mit Tuberkulin gelingen wird, die tuberkulösen Individuen eines Rinderbestandes zu

erkennen. Bekanntlich ist in einer Reihe von Versuchen die Thatsache hervorgehoben worden, dass auch völlig gesunde Tiere auf Tuberkulinimpfung Reaktion gezeigt hätten. Von vornherein war schon die Vermutung gerechtfertigt, dass wenigstens bei einem Teil dieser als völlig gesund bezeichneten Tiere doch an irgend einer versteckten Stelle ein tuberkulöser Herd vorhanden gewesen sein möge, und in der That zeigte die subtile Untersuchung sämmtlicher Lymphdrüsen an den Eingangspforten in einer umfassenden Versuchsreihe, dass solche unerwarteten Reaktionen auf geringe und streng lokalisirte Herde im Körper zurückzuführen waren.

Bei der grossen Anzahl von Kühen, welche an evidenter Tuberkulose leiden, 10·36 %, und dem nicht unbedeutenden Procentsatz, welcher nur Tuberkulose der einen oder anderen Lymphdrüse aufweist, erscheint es vorläufig geradezu als *ein Ding der Unmöglichkeit, die Milche aller Tiere vom Consume auszuschliessen, welche auf eine Tuberkulininjection reagiren.* Wir können nicht unvermittelt auf ein Drittel der täglichen Milchmenge Verzichtsleisen, ohne den Preis dieses unentbehrlichen Nahrungsmittels ganz ungebührlich in die Höhe zu schrauben und den Genus derselben den ärmeren Volksschichten zu verringern oder gerade zu unmöglich zu machen. Die allgemeinste Anwendung der Tuberkulininjectionen zur Eruirung der tuberkulösen Stücke in den Milchwirtschaften ist im höchsten Grade wünschenswert und muss staatlich angeordnet werden, sobald über die bereits angestellten Versuch ein abschliessendes Urteil gefällt werden kann, namentlich, auch darüber, ob bei Anwendung einer Minimaldurchschnittsdosis die Intensität des Fiebers je nach Ausbreitung und sonstiger Beschaffenheit des tuberkülosen Processes schwankt. Die Ausmerzung der durch Tuberkulin ermittelten tuberkulösen Tiere kann selbst bei staatlich geregelter Entschädidung nur allmählich geschehen, bis wir uns einen gesunden Viehstapel herangezüchtet haben werden. Dagegen ist die Separirung der auf Tuberkulin reagirenden Kühe und ihre Ausschliessung von der Nachzucht jetzt schon möglich. Ausserdem wären die Besitzer anzuhalten, diejenigen Tiere möglichst bald auszumerzen welche ausser dem Ergebniss der Tuberkulininjection noch anderweitige, den Verdacht bestärkende Symptome der Tuberkulose erkennen lassen. *Die Milch aber der lediglich auf Tuberkulin reagirenden Kühe kann, wie ich glaube, nach vorherigem Aufkochen oder Sterilisiren nach einem bewährten Verfahren unbedenklich in den Verkehr gegeben werden.* Denn schon das einfache Aufkochen genügt nach den Feststellungen von Bang zur sichern Tötung der Tuberkelbacillen in der Milch.

Es wird sicherlich nicht an Stimmen fehlen, welche die vollkommene Ausschliessung der Milch der auf Tuberkulin reagirenden Kühe verlangen. Von diesen wird aber völlig verkannt, dass es bereits einen gewaltigen Fortschritt in der Milchhygiene bedeutet, wenn die möglicherweise virulente Milch tuberkulöser, beziehungsweise der Tuberkulose verdächtigen Kühe, welche früher ahnungslos als völlig tadellose Milch genossen wurde, nach vorheriger Unschädlichmachung in den Verkehr gebracht wird. An Käufern derartiger Milch wird es

nach öffentlicher Klarlegung der Verhältnisse durch die Behörden ebenso wenig fehlen wie an Käufern der gekochten Milch von aphthenseuchekranken Tieren und des Fleisches von tuberkulösen Tieren, wenn die Milch nur zu einem etwas geringeren Preise abgegeben wird, als diejenige nicht tuberkulöser Tiere. Es muss aber behördlicherseits dafür gesorgt werden, das die Kochung der Milch, welche von tuberkuloseverdächtigen Kühen stammt, ebenso durchgeführt wird, wie in Deutschland bei der Aphthenseuche. Die Schwierigkeiten, welche sich der Ausführung eines solchen Verfahrens in den Weg stellen, dürfen bei der hervorragenden hygienischen Wichtigkeit derselben keinnen Grund abgeben, dasselbe überhaupt unausgeführt zu lassen.

Als letzte Gruppe von Erkrankungen der Milchtiere mit Hinsicht auf übertragbare Krankheiten kommen *die Entzündungen des Euters* in Betracht. Die Aetiologie der Euterentzündungen ist keine einheitliche, und in Folge dessen muss auch bei den verschiedenen Formen der Mastitis das noch gewinnbare Produkt verschieden beurteilt werden. Klinisch kann man die Euterentzündungen einteilen in phlegmonöse, katarrhalische, abscedirende und gangränescirende; ausserdem muss noch die bereits besprochene tuberkulöse sowie die aktinomykotische Mastitis unterschieden werden. Letztere kommt beim Schweine ziemlich häufig vor ; beim Rinde dagegen zählt sie zu den seltensten Ereignissen. Der Ausschluss der Milch bei letztgenannter Erkrankung muss aus naheliegenden Gründen verlangt werden, wenn auch gleich weder eine Uebertragung der Aktinomykose auf den Menschen noch nicht beobachtet worden ist, noch für überhaupt wahrscheinlich gehalten wird. Von der phlegmonösen Euterentzündung können wir hier absehen, weil dieselbe in der Subkutis und im interacinösen Gewebe sich abspielt. Grosses Interesse aber bieten die übrigen Formen der Mastitis. Bei der katarrhalischen Form fand Kitt regelmässig die sogenannten Mastitisbakterien. Bang konnte bei einer chronischen ansteckenden Euterentzündung einen besonderen Streptococcus als Ursache ermitteln; aus anderen entzündeten Eutern züchtete er Streptococcen, Bicoccen, Staphylococcen und Bacillen, welche in die Milchcysterne gebracht wiederum eine Entzündung des Euters auslösten. Nocard und Mollereau, sowie Hess und Borgeaud stellten als Ursache einer sehr ansteckenden chronischen Mastitis, bei welcher die Milch sauer aus dem Euter kommt und Atrophie und Agalaktie die schliesslichen Folgen sind, Streptococcen fest, welche nach Kitt von den Bang'schen verschieden zu sein scheinen. Experimentell konnte parenchymatöse Mastitis hervorgerufen werden durch Injection der Bacillen der blauen Milch, der Hühnercholerabakterien, der Drusestreptococcen in die Milchcysterne. Staphylococcus pyogenes aureus erzeugte nur eine vorübergehende Veränderung mit Schwellung des Euters. Eine beträchtliche entzündungserregende Wirkung aber folgte der Injection von Botryococcus ascoformans sowie derjenigen des Bacillus euteritidis, Gärtner. Wenn nun auch nur vielleicht von den letztangeführten Bakterien eine pathogene Wirkung für den Menschen mit Sicherheit angenommen werden kann, so muss doch wegen der

Schwierigkeit der speciellen Diagnose im Einzelfalle und weil die Unschädlichkeit der übrigen Bakterienarten für den Menschen nicht erwiesen ist, die Ausschliessung aller Milch von euterkranken Kühen vom Consume verlangt werden. Die Milch darf erst dann wieder zum Consume zugelassen werden, wenn die Entzündung völlig abgeheilt und die gemolkene Milch frei von Caseïngerinnseln, Blut oder Eiter ist.

Hiermit wären die Hauptquellen der gesundheitsschädlichen Milch und die dagegen anzuordnenden Massregeln besprochen. Schliesslich kann aber noch die *bereits gemolkene* Milch durch zufällige Berührung mit infectionskranken Personen oder mit Gegenständen, welche durch pathogene Keime verunreinigt sind, inficirt werden. Durch zahlreiche Beobachtungen is dieser Infectionsmodus, welcher bei der Eigenschaft der Milch als eines ganz vorzüglichen Nährbodens leicht verständlich ist, als erwiesen zu betrachten. England gebührt das Verdienst, auf diese Gefahr zuerst aufmerksam gemacht zu haben; später wurde auch aus Dänemark, Deutschland und Holland über ähnliche Fälle berichtet. Alle diese Fälle haben das Gemeinsame: In einer Milchwirtschaft tritt ein Fall einer Infectionskrankheit auf, plötzlich folgen " explosionsartig " Erkrankungen, dem stärkeren Milchconsume entsprechend, namentlich der Frauen und Kinder und schliesslich wird die Krankheit durch das Verkaufsverbot der schädlichen Milch coupirt. Bezüglich der Einzelheiten verweise ich auf die vorzüglichen Literaturstudien in den bereits erwähnten Arbeiten. Dort ist auch näher begründet, dass ein Teil der Beobachtungen, welche auf die Übertragung von Infectionskrankheiten durch Milch Bezug haben, nicht als beweisfähig angesehen werden kann. Als festgestellt wird aber angesehen die Übertragungsmöglichkeit für Cholera und Typhus, als immerhin wahrscheinlich für Diphtherie und auch für Scharlach. Die Möglichkeiten für eine nachträgliche Infection der von gesunden und normal gehaltenen Kühen stammenden Milch sind kurz folgende: Einstreu von Stroh aus Krankenbetten, infectionskrankes oder im Reconvalescenzstadium befindliches Stallpersonal, mit pathogenen Keimen beladenes Spülwasser und endlich die Aufbewahrung der Milch in Räumen, in welchen sich infectionskranke Menschen befinden. Die Vorbauung gegen die nachträgliche Infection der Milch ergiebt sich von selbst. Es muss die Einstreu von Stroh aus Krankenbetten, die Verwendung infectionskranker und reconvalescenter Menschen in der Milchwirtschaft verboten, ferner dafür Sorge getragen werden, dass nur Wasser zum Spülen der Milchgeräte verwendet wird, welches aus guten, einer Verunreinigung unzugänglichen, Brunnen stammt. Beim Ausbruch einer Infectionskrankheit müssen die Kranken streng separirt werden; wo dieses nicht durchführbar erscheint, ist die Milchwirtschaft während der Dauer der Krankheit u schliessen. Die Aufbewahrung von Milch in Schlaf- und Wohnräumen ist mit Rücksicht auf die beregte Gefahr im Allgemeinen zu verbieten.

Zum Schlusse wäre noch des Uebergangs schädlicher Metalle, Blei, Kupfer und Zink aus den Transportgefässen und des Verbotes der

Verwendung von Gefässen zu gedenken, welche diese Metalle führen.

Die im Vorstehenden begründeten Forderungen müssen für den Verkehr mit *gewöhnlicher Marktmilch* aufgestellt werden. Eine besondere Würdigung verlangt, wie hier nur der Vollständigkeit halber noch angeführt sein soll, der *Verkehr mit Kinder-und Kurmilch.* Es ist im Interesse einer gedeihlichen Entwickelung der künstlich ernährten Kinder im höchsten Grade wünschenswert, einen Teil der Milchwirtschaften zur Production dieser sogenannten Vorzugsmilch zu veranlassen. Von solchen Producenten muss aber ausser den genannten Vorbeugungsmassregeln gegen gesundheitsschädliche Milch noch die strenge Beachtung besonderer Massregeln verlangt werden, und zwar Fütterung der Kühe entweder mit guten Grass oder Heu, allenfalls von Zugabe von Mehl, Kleie. Von technischen Rückständen dürfen nur erwiesenermassen unschädliche, zum Beispiel Bierträber, als Beifutter Verwertung finden. Das Heu ist nach Soxhlet zur Vermeidung des für die Haltbarkeit der Milch so sehr nachteiligen Heustaubes wo möglich angefeuchtet zu verfüttern. Im Uebrigen ist grösste Sauberkeit der Ställe, Reinigung der Euter sowie der Hände des Melkers vor dem Melken und peinlichste Reinhaltung der Milchgeräte dringend zu fordern. Ausserdem ist die Milch nach dem Melken aus den allgemein bekannten Gründen mittels besonderer Kühlvorrichtungen abzukühlen und wo immer möglich sterilisirt, sonst aber mit dem geringsten Zeitverlust den Consumenten zu überbringen.

## DISCUSSION.

**Dr. Leonard Pearson** (Philadelphia) observed that Dr. Ostertag was strongly of opinion that it was decidedly the duty of the State to see that only pure milk entered the market. The consumer was not in a position to guard himself against the manifold dangers which attended the consumption of milk, which might contain the most harmful ingredients in spite of white colour and sweet taste. Such milk could only be banished from the market if the milk-supply were controlled by Government officials. Dr. Ostertag urged that only pure milk should be tolerated in the market—that was, milk obtained with the greatest cleanliness from healthy animals, possessing normal physical qualities and a certain degree of strength. For sanitary reasons the following kinds of milk must be excluded from the market:—(1.) Milk which, without being necessarily prejudicial to health, was peculiar in colour, taste, or consistence [nauseous milk]; (2.) All milk that was prejudicial to health, or which was suspected, on good grounds, of being so. To the first group belonged colostral milk, blue, red, and yellow milk; also, slimy, thready, bitter, salt, as well as abnormally smelling milk, and milk that had been made impure by mud or other substances. The milk of animals that had been fed on poisonous fodder, or that had been treated with certain medicaments, and of those suffering from tuberculosis, malignant pustule, cow-pox, foot-and-mouth disease, or of those generally ill in consequence of

some process inducing ulceration or ichor, must be regarded as prejudicial to health. The possibility of milk being of a hurtful nature was suggested in all the other feverish ailments common to milk-yielding animals, as also by the different forms of inflammation of the udder. Again, milk which had already been drawn might become infected by immediate contact with sick persons (typhus fever, cholera, &c.), or through being kept in rooms where such persons were. Finally, through being carried in unsuitable (metal) vessels, injurious subtances might find their way into the milk. Dr. Ostertag made a number of recommendations for guarding against these dangers, the most important of which was that all dairy farms should be licensed.

**Mr Francis Vacher** (Medical Officer of Health for Birkenhead) considered that Dr. Ostertag's recommendations for securing a wholesome milk supply were excellent. Dr. Ostertag's opening remark that most countries had paid but slight attention to the sanitary question of the milk supply was certainly less true of this country than of others. The Sale of Food and Drugs Acts, when due effect was given to them, protected the public from skimmed and watered milk. The regulations made by Local Sanitary Authorities, under powers given them by Privy Council Orders, enabled such authorities to register, inspect, and keep free from contamination the milk offered for sale locally. The Contagious Diseases (Animals) Acts secured the due notification of Foot-and-Mouth Disease and of some other epizootics. Finally, there were powers given by many local Acts requiring that outbreaks of human disease in dairies, milk-shops, &c. should be reported. Three things were required if the public of this or any country were to be safe as regards their milk-supply:—(1.) Registration and licensing of all milk farms and milk-shops; (2.) Efficient inspection (not only by medical officers of health and their subordinates, but by veterinary surgeons) of all such farms and shops; and (3.) Education of the public to appreciate the security offered to themselves by boiling the milk.

**Professor Brown, C.B.** (London), said some speakers on the previous day had complained, not without good reason, that the cow was rather hardly used in consequence of the tendency, which was greatly increasing, to ascribe all kinds of diseases to the consumption of milk derived from that animal. But no one was disposed to question the undoubted fact that milk was subject to a very large amount of contamination. Without reference to any specific malady, and altogether distinct from the question of the possibility of the cow suffering from a disease which would confer upon the milk the power of producing some other disease in the human subject, stood the distinct fact that milk was contaminated by dirt from the hands of the milkers and by the excreta of the animal. Milk was also contaminated by inflammatory products in nearly every disease to which the cow was subject. Only recently h had succeeded in separating thirteen organisms from the milk of a perfectly healthy cow, collected carefully under proper antiseptic conditions. The presumption was that none of these organisms was injurious. Milk was also contaminated by septic inflammation of the udder in the form of mammitis, and they had evidence that its contamination with the morbid product in this disease might take place owing to ignorance on the part of the milker. He instanced a case in which a milker milked into the pail a quarter of the contents of the udder, from which came nothing but pus. Apparently his impression was that the somewhat brilliant yellow tinge and thick character of the pus imparted a certain

degree of colour and quality to the rest of the fluid, a sample of which showed that it was full of septic organisms. Milk was also contaminated with specific diseases, such as pleuro-pneumonia, tuberculosis, and foot-and-mouth disease. He was sorry to say that the admirable regulations referred to by Mr. Vacher were by no means general throughout the country. There was a large number of towns in which there were either no regulations at all or no attempts made to enforce such regulations as did exist. The consequence was that cows were milked with dirty hands, their udders were dirty, and the milk was milked into dirty pails. Something in the way of general regulations was required. When the Privy Council had charge of the matter, it was their experience that it was very difficult to induce local authorities to take advantage of their powers: out of some 500 authorities not more than 50 or 60 made any regulations at all, and it was on this account that it was deemed advisable to transfer the powers under the Contagious Diseases Act, section 34, to the Local Government Board, who had machinery for dealing with this subject which the Privy Council did not possess. Since this transfer it had not been his duty to make inquiries, but he was afraid that there were not many towns in which the arrangements were so satisfactory as those made by Mr. Vacher, and carried out under his supervision. Moreover, for the effectual enforcement of any regulation, it must be admitted that a system of compensation to the dairyman would be absolutely necessary. It was a serious hardship for a man who had a small business that his whole trade should be stopped by the sanitary inspector, without any authority having the power to give him a shilling in compensation. That of course could only be remedied by legislation. Mr. Vacher had referred to the desirability of boiling the milk. This was a point upon which distinct prejudice existed. Little children had a strong objection to it, and their objections were accepted by their parents. With reference to this subject, he quoted from the *North British Agriculturist* the following curious paragraph which he gave for what it was worth and as of interest to the Congress .—

"The results of Dr. Freundenreich's experiments, as now published in the *Annales de Micrographie*, are of first rate importance. He finds that the cholera bacillus, if put into milk drawn fresh from the cow, dies in an hour, and in five hours if put into fresh goat's milk. The bacillus of typhoid fever takes twenty-four hours to die in cow's milk, and five hours in goat's milk. Other microbes suffer like fate in varying periods. By this showing, fresh milk is a bactericide or killer of disease-causing micro-organisms. But Dr. Freundenreich's researches go yet further. He finds that milk maintained for an hour at a temperature of 55 degrees (131 F.) loses its power to kill microbes—a statement which is interesting in face of the common teaching, which makes the purification of milk depend on its being boiled. Again, the microbe-killing properties of milk becomes weaker the older it gets. Cow's milk after four days, and goat's milk after five days cease to have any effect upon micro-organisms. The conclusions, at any rate, are altogether in favour of the consumption of fresh milk."

He had not the slightest evidence in support of these statements, which it was possible would be refuted later on.

**Professor E. Perroncito** (Turin) dit:—Des observations faites sur le lait et le beurre, et communiquées par le Dr. Binsaferro à l'Académie Royale et Nationale Vétérinaire de Turin, ont démontré la présence du bacile de la tuberculose vraie et le microbe de la pseudo-tuberculose. En Italie, à Turin, nous avons un règlement qui prescrit des visites aux vacheries pour éliminer du commerce tout le lait des vaches malades,

spécialement le lait des vaches tuberculeuses. Une vache tuberculeuse a été vaccinée et saturée par le virus du charbon. Ensuite elle a semblé améliorée. J'ai soumis à la même vache pour prendre le lait un veau de deux mois parfaitement sain. Ce veau a pris la tuberculose et les symptômes de la maladie se sont manifestés après deux mois environ. Aux mammelles de la vache, il n' y avait aucune lésion locale de la tuberculose.

**Dr. Henry E. Armstrong** (Medical Officer of Health for Newcastle-on-Tyne) said that in Dr. Ostertag's reference to the spread of typhus by means of milk, he of course did not mean the disease which the English termed typhus, but enteric fever (or abdominal typhus, as it was termed on the Continent). The supervision of the sanitary arrangements of dairies—especially country dairies supplying milk to towns—should be under the full control of the Sanitary Authorities of those towns. Within a year, the speaker had had experience of an outbreak of enteric fever in Newcastle, associated with milk supplied from a country dairy, at which the water for refrigerating the milk had been drawn from a well beneath the farm-yard dung-midden. He had also learnt that on one occasion the tube of the refrigerator had come off, and had allowed several gallons of the foul water to get into the milk. It was desirable to have compulsory notification of certain diseases of cattle not included in the provisions of the Contagious Diseases (Animals) Acts, viz., tuberculosis and parturient fever. This notification should be made the duty both of the veterinary surgeon and of the cow-keeper, similarly to the dual system of notification of human infectious diseases. The milk of recently calved cows, containing colostrum, should not be allowed to be sold for food. He (Dr. Armstrong) had for many years advocated that the minimum of space allowed for each cow in a cowhouse (at least in town) should be 800 cubic feet. To some extent compensation to dairymen for milk condemned was desirable; but it should be remembered that small dairies in towns, were, as a rule, kept in a grossly insanitary condition, which was seldom the case in the large dairies. Hence some distinction should be made. He claimed the credit of having been the first to advocate that Local Sanitary Authorities should have the power to compel the owners of dairies to give lists of their customers in connexion with outbreaks of infectious disease. This power was obtained for Newcastle as far back as the year 1882. The advocacy of the boiling of suspected milk was, like the advocacy of thorough cooking for measly pork, most objectionable. Another and far better course was necessary, viz., the prevention of the possibility of the infection of the milk, or the destruction of all milk not known to be free from suspicion.

**Professor Walley** (Edinburgh) asked wherein the great objections lay to the use of colostral milk? Did not the mother allow her child to suck immediately after birth? Was it not a fact that if calves did not get colostral milk, they suffered from derangement of the digestive organs, and was it not a further fact that thousands of gallons of colostral milk (beestings) were sold in England for the purpose of making custard? In reference to cleanliness of fodder and water, Professor Walley pointed out that some dairymen allowed brewers' grains to lie until they became decomposed; this he considered a very reprehensible practice, such food often producing the worst forms of indigestion in the cow. As to polluted water he directed attention to a case that had come under the observation of Professor Law of the Cornell University, in which the milk of cows drinking from a polluted well was found to contain microbes

identical with those found in the drinking water. Cleanliness in dealing with dairy cows could not be too much insisted on. All cows' udders and teats should be washed prior to milking, but as a rule it was difficult to get dairy people to be cleanly, and they often pointed out the healthy condition of themselves and their milkmaids as proof that dirt did no harm. In reference to boiling, he quite agreed that it did alter the character of the milk, and he suggested that it should be subjected to a similar process to that to which Swiss milk was subjected. Milk so treated, he could say from his own experience, was admirable for children. But he pointed out that "one cow" milk for children was highly dangerous to the child, as it might be drinking undiluted tuberculous milk. Mr. Vacher had referred to milk from cows suffering from pustular fever; he, the speaker, had never seen such a condition of things unless mammitis had been set up. Professor Brown stated that milk from pleuro-pneumonia cows was dangerous: he (Professor Walley) had never observed any change in such milk. He was pleased to gather from Professor Brown's remarks that the recognition of the principle of compensation was extending. He quite agreed with Dr. Armstrong that tuberculosis should be scheduled as a contagious disease, but he did not understand why he should desire that parturient fever—if he meant from that term so-called milk fever—should be made a notifiable disease, seeing that the milk was not secreted in that affection; in fact, its total suppression was one of its most pronounced clinical phenomena. Professor Walley agreed with Dr. Armstrong in reference to the cubic space of 800 feet per cow being insisted on, and as a matter of fact, it was one of the regulations in force in the Edinburgh dairies.

**Dr. M. K. Robinson** (Dover) described an epidemic of typhoid fever which he investigated twenty-four years ago, when he was led to the conclusion that the milk was the vehicle of the specific poison, and that it became infected by absorption and not through contaminated water. Since then he had looked upon milk as a vehicle of the poison from human infection and not from diseased cows. With reference to Professor Brown's remarks upon the view that milk had been regarded as a microbe killer, and therefore not likely to convey disease, his experience of many epidemics clearly produced by infected milk could not have occurred if milk possessed such power. An epidemic of aphthous fever which occurred in Dover some few years ago, was clearly traced to the consumption of milk from cows suffering from the same disease, and this outbreak proved that disease occurring in milch cows could be produced in human beings. He thought too much stress had been laid upon microbes and not enough on the products of the microbes, which would explain not only the different appearance of the microbes examined, but also the diversity of symptoms observed in human beings, during the prevalence of any special epidemic.

**Dr. George Fleming, C.B.** (London), drew attention to the confusion existing as to the meaning to be applied to the term "milk fever." Grave wrong was done to owners of cows through this confusion. In fact, under this designation two widely different maladies were included: "Milk or Parturient Fever" and what had been named "Parturient Apoplexy." The first was a septic fever, and the milk—if there were any—should not be allowed as food, neither should the flesh, as serious symptoms might supervene in those who consumed them. The distinction should be observed, as, in the other disease, "Parturient Apoplexy" there was no danger, unless the animal had received medicines which

would render the flesh or milk hurtful. The observations of Professor Perroncito deserved notice, not only with regard to the regulations in force in Turin, but more especially with respect to the discovery that butter made from the milk of tuberculous cows would communicate tuberculosis. This disease was so common and so serious that it was well to know all the sources by which it might be extended.

**Dr. Edward Sergeant** (Medical Officer of Health for Lancashire) called the attention of the meeting to the danger of "maturing" cheese in the bedrooms of small farms. This practice was not uncommon in some districts, and he had known cases of diphtheria and other infectious diseases treated in rooms used for the storing of cheese. It was obvious that sanitary authorities should insist on dairy farms being provided with suitable buildings for the proper conduct of their trade without danger to the public health. The regulations under the Dairies and Cowsheds Order, if adopted at all, were not carried out as they ought to be; many small farms were very dirty, and the conditions surrounding the cattle did not conduce to the health of the animals or the purity of the milk. Instead of small sanitary authorities being empowered to supervise farms, it would be more desirable if County Councils were entrusted with this supervision. Compensation should be given, as suggested by Professor Brown, to farmers or dairymen whose sale of milk under the Infectious Diseases Prevention Act of 1890 might be interfered with, and it was unfortunate that this provision was not included in so recent an Act. He agreed with the observations which had been made with respect to the notification to the sanitary authority of certain diseases amongst cattle. The notification should be dual: *i.e.*, by the duty being imposed on both the dairyman and the veterinary surgeon.

**The President,** in closing the discussion, remarked that they had learned that day the necessity of stringent regulations being imposed on all who sold milk. Nobody would wish to curtail the sale of milk or to prevent it being sold at the least possible price to the population, especially in towns; but at the same time great care must be taken that in the supply of that milk disease was not distributed. They had only to look at some of their large dairy companies who went to the source of the evil, viz., the farms where the milk was produced, and who imposed stringent regulations as to cleanliness and declaration of infectious diseases upon those who looked after the milk. If private enterprise could do that, with a little more power from the Legislature the most stringent regulations might be carried out, without detriment to the trade.

## The Inspection of Meat with reference to the Spread of Disease.

BY

D. E. Salmon, D.V.M., Chief of the United States Bureau of Animal Industry.

The inspection of meat, as a sanitary measure, has been attracting more and more attention with each succeeding year, and the scientific questions bearing upon this subject have been investigated wit much vigour and

patience. Results have been obtained of great importance, and while we undoubtedly have much yet to learn, we have reached a point where there can no longer be reason for the radical differences of opinion which have caused so much discussion in times past. It is not my intention, however, to enter into the details of the researches to which reference has just been made, as these are already known to the scientific world. I prefer to treat the subject in a more general manner, and to suggest certain topics which may still be discussed with much profit.

The meat inspector's duties are not confined to condemning the carcasses of animals which are affected by a disease which may actually be transmitted to the consumer, but he must act with equal energy when confronted by conditions which may be accompanied by the unusal production or the retention of noxious compounds within the tissues. Further than this, in the high development of civilisation at the present day, the sensitive consumer expects to be protected from the use of animal food which, though not positively harmful, is obtained from animals whose condition would be repugnant or disgusting to him were he to see them before their slaughter.

Conscientious meat inspection is no easy task, for a disease which may not be communicable may cause the presence of a poison which would be as injurious as the malady itself, and a disease or a condition which is neither communicable nor known to be in any way harmful, may be of such a nature as to be repugnant to our cultured tastes, though it would bring no hesitation to the minds of savages. The meat inspector must, therefore, be a man of science, who can tell from the lesions which he sees the nature and the invisible effects of the disease which produced them, and he must be sufficiently a man of the world to condemn, for decency's sake, certain carcasses which to the best of our knowledge wou'd not be injurious to the consumer. He must also be a man of discretion, because there is no hard and fast line where a disease in its development becomes injurious or a condition repugnant.

The methods according to which the meat inspector works are of the greatest importance in determining the value of his services. It has been customary in many places to rely upon an examination of the carcass after it has become cold, or of the meat as it is exposed for sale. Although the experienced inspector becomes extremely expert, and can detect conditions which most of us would fail to observe, this method is wanting in accuracy, and is insufficient to detect all diseases which should subject a carcass to condemnation.

Having lately been engaged under the direction of Secretary Rusk, of the Department of Agriculture, in inaugurating a national meat inspection service in the United States of America, in accordance with a recent Act of Congress, I have had occasion to give considerable attention to this subject. For our inspection, it has been decided to be essential that an inspector should see every animal before it is killed, and should be present to examine the viscera when removed from the carcass.

With a single animal such an inspection is simple enough, but when we enter an abattoir with a killing-floor acres in extent, where from two to four thousand beeves are slaughtered daily, the problem suddenly

appears complex, and can only be solved satisfactorily by proceeding systematically, and by a division of labour.

The difficulties will be appreciated by anyone who visits the abattoirs of the foreign animals wharves at Deptford, in London, or the immense packing houses at Kansas City, Omaha, and Chicago, in the United States. An inspection of these great establishments, however, while difficult, is by no means impossible. With a sufficient force of inspectors properly stationed, every animal can be examined as it goes to the abattoir, and every carcass can be seen while the butcher is removing the viscera.

According to the system adopted in the United States there are outside inspectors and inside inspectors. The outside inspectors are not expected, of course, to feel the animals' pulse, or take their temperature, or auscultate their lungs. But they are expected to see if the general appearance indicates health, to reject animals which have been injured during transportation, or which have external swellings or abscesses likely to affect the system of the animal. In case of doubt they notify the inside inspectors, who give the animal in question particular attention. The inside inspectors examine the viscera of every animal, and as both inside and outside inspectors must be veterinarians, they are able to decide at once as to the nature of any disorder which they observe, and its effect upon the carcass of the animal.

What are the conditions which justify an inspector in condemning a carcass and ordering its destruction? Surely we cannot limit him to diseases which are with certainty communicable to man through the consumption of affected meat. Should we do so, meat inspection would be regarded by the majority of consumers as an unmitigated farce.

Take as an example the class of diseases of which rabbit septicæmia is a type, and which includes, besides the malady just mentioned, fowl cholera, swine plague, a form of pneumonia in bovine animals, and the disease described by the Germans under the name of *wildseuche*. This class of diseases is widely distributed over the world, it affects nearly all species of meat-producing animals, it is accompanied by constitutional disturbances and tissue changes of a very marked character. And yet, so far as we know, these maladies are not communicable to man, nor am I aware of any special evidence demonstrating that the meat affects the health of the consumer in any way.

M. Reynal says in his article on fowl cholera in the veterinary dictionary of Bouley and Reynal, *Nouveau Dictionnaire de Médecine, de Chirurgie et de Hygiéne Vétérinaires, Tome III, p. 658:* "A labouring man at the school took, in 1851, all the fowls which died of the inoculated or spontaneous disease; they were used for the nourishment of himself and his family of five children; none of these were incommoded by this alimentation.

"Like remarks were made in 1789 by Baronio in Lombardy; in 1832, by Grognier; the dead fowls were consumed without the least inconvenience.

"In 1851, when this epizootic had invaded nearly all the farms in the suburbs of Paris, masters and servants consumed without becoming

incommoded thereby the fowls which died or which were killed during the course of the disease.

"Daily, these fowls were purchased by the merchants from the farmers, and sold either to their customers or in the market of *La Vallée;* this sale no doubt was made without the knowledge of the authorities, but we can affirm, in the most absolute manner, that the meat occasioned no derangement in the health of the persons who made use of it.

"The innocuousness of the meat of fowls affected or dead of this epizootic malady is demonstrated by facts so numerous and so authentic, that the authorities ought to tolerate the sale of them."

Various authorities have also advised against the condemnation of the carcasses of bovine animals which were affected with the contagious pleuro-pneumonia. In France the sale of the carcasses of animals slaughtered with this disease is left to the discretion of the veterinary inspector; and, in Great Britain, if Professor Walley is correct, it is the "universal rule to pass them as marketable and innocuous if they present "no departure from natural conditions—the affected portions of the "pleuræ being removed by stripping, and, in bad cases, the portion of "the fore quarter contiguous to the pleuritic lesion, or even the whole "of the quarter, being retained." In the United States, during the work of eradicating pleuro-pneumonia, the carcass of every affected animal was destroyed.

It has also been a common practice in European countries, during outbreaks of foot-and-mouth disease, to slaughter animals for food even when the fever of this disease was at its highest point. This is shown by a short quotation from Prof. Walley's recent work on meat inspection. He says, "during the prevalence of an epizootic of this disease, thousands of animals, while labouring under its effects, are slaughtered, and their flesh used for human food. . . . . So far as I am aware there is no instance on record of the transmission of the disease to man through the medium of the flesh of affected animals. . . . In reference to the use of the affected parts as articles of human food, no difference of opinion need exist; if the feet, the tongue, or the udder, are the seat of local lesions, they should be destroyed, and the same applies to the tripe; but I cannot agree with those who would condemn *in toto* the head, the heart, and similar organs," pp. 118, 119.

These quotations are good illustrations of the fact that a contagious disease, having a virus of extraordinary virulence for certain species of animals, may not render the flesh of such animals actually harmful to the consumer. The carcasses of animals which have died of such diseases, or those which have been slaughtered after the first symptoms have appeared should, however, in my judgment be rejected, or the consumer should be informed of the malady from which the animal had suffered.

With us, in the United States, an animal which is sick, no matter what the disease, is considered as unfit for food, and our people would not knowingly tolerate an inspection which allowed the carcasses of such animals to go upon the market. From having for a long time an abundant supply of cheap meat, our people are accustomed to choose the best cuts from the best animals, and they are extremely impatient in

regard to any policy which has a tendency to allow a food product to be placed upon sale which they would reject were they to know all the facts concerning its origin.

An animal which has a decided elevation of temperature from any cause, whether from the effects of a contagious disease, or from an ordinary fever, or from a severe injury is, according to our standard, unfit for human food. An animal in an advanced condition of pregnancy or a parturient animal, as well as unborn and recently born animals, are pronounced unfit for food. For the same reason, animals with large abscesses, whether these affect the general condition or not, and those affected with actinomycosis and tuberculosis, whether generalized or not, are all condemned.

In the condemnation of cattle affected with actinomycosis, we have probably gone farther than any other country, since beeves in perfect condition have been condemned when they only presented a tumour on the maxillary bones the size of a walnut. It is in defining the exact point in the progress of such conditions, where condemnation is called for, that we find the greatest difficulty in meat inspection. If a suppurating tumour on the jaw, 10 inches in diameter, calls for condemnation of the carcass, why should not one which is eight inches, or even six inches in diameter, and, if these are condemned, what shall we say of those which are five, four, three and two inches only? Where shall the line be drawn? This is a question which might well be discussed at considerable length during the sessions of this Congress, for actinomycosis is becoming the disease most frequently encountered in the large stockyards of the world.

Again, should we allow a female animal to be slaughtered for human food the same day that it has given birth to its young, or at a time when parturition is evidently near at hand? If not, how many days shall the owner be compelled to maintain the animal before it is slaughtered, and at exactly what age is the offspring to be considered fit for food? These are problems which confront the meat inspector; they are essentially sanitary problems and of interest the world over.

The United States Government is now attaching a numbered card, bearing the words, "inspected meat," and the signature of the Secretary of Agriculture, to each quarter of beef that has been inspected. This card is attached by means of a wire and lead seal. To identify inspected meat in boxes, packages, or cans, a meat inspection stamp is affixed to each case. All meats covered by these stamps are the product of animals that have been inspected before and at the time of slaughter, and, in the case of pork, specimens from each carcass have been examined microscopically and pronounced free from trichinæ.

Sanitarians no doubt will feel like inquiring as to the necessity of an enormous inspection service to search for a microscopic worm, which is killed by a comparatively low temperature, and even by the ordinary processes of curing the meat. When meat is thoroughly cured or cooked there need be no fear of this parasite. We have in the United States a population of about 64 millions of people, which consumes more pork *per caput* than is eaten elsewhere in the world. Many of our people

eat ham which has been salted and smoked, but not cooked. And yet trichiniasis is an exceedingly rare disease, and in every case that I have investigated, and this includes most which have occurred in recent years, it was contracted from eating raw meat that had not been salted. The most reliable protection from this parasite is, therefore, to have all varieties of swine-flesh either salted or cooked before they are eaten.

I contend that salting and cooking is a better protection than microscopic inspection, because we have no trichiniasis in the United States from meats so treated, nor have any cases been caused in Europe by the millions and billions of pounds of salted pork that we have exported. On the other hand, there have been frequent and terrible epidemics of this disease in Germany, from the time microscopic inspection was instituted, about 1875 or 1876, until the present day, and many, if not all, of these epidemics resulted from inspected pork.

The United States, however, is a meat-exporting nation. It recognises the fact that its meat inspection system has become a matter of international interest, and is determined to make it so rigid and comprehensive that it cannot be excelled by the most progressive nation in the world.

We have not been entirely lacking in inspection in the past. We have had Board of Trade inspections, Municipal inspections, and State inspections, and we now have in addition the National inspection. Much of the meat will in future be examined by all four of these classes of inspectors, and, if there is any value in inspection, the consumer should hereafter eat American meats with a feeling of great relief and confidence.

The great interest in scientific research at the present day, the rapidity with which the literature of all nations is circulated, the numerous occasions on which scientists of various countries meet in international congresses, are bringing about greater uniformity of opinions, and a clearer idea of the reasons for national or individual variations from our standard. Admitting this, there can be no doubt that the sanitary regulations of different countries will continue to approach more nearly to a common standard, until the people of any one nation can visit the people of any other nation, live in their dwellings, mingle with their crowds, and eat their food without fear of sacrificing their health by so doing.

With all the energy of recent scientific investigation, it is a fortunate outcome that so staple an article of food as meat should prove to be less frequently a medium for the dissemination of disease than a few years ago we had reason to believe. Even tuberculosis cannot be distributed in this way to the extent we once supposed, or we should find it more frequently located in connection with the digestive than with the respiratory apparatus. And the products of the multiplication of disease germs are not as dangerous when taken into the stomach as we once believed, or those who consume the flesh of animals which have died of or were affected with fowl cholera, pleuro-pneumonia, and foot-and-mouth disease, could not do this with the impunity which has been proved to be possible. Indeed I had occasion to show, several years ago,

that the products of certain pathogenic bacteria failed to produce the same specific effects when administered by way of the stomach that they did when injected hypodermically. Dr. Koch afterwards made the same observation in regard to the use of the products of the bacillus of tuberculosis. We may feel assured, therefore, that some of these products, at least, are so changed by the processes of digestion as to lose their peculiar properties.

No doubt there is much yet to learn in regard to the communicability of animal diseases, and even more to learn about the effects of bacterial products; but it is a relief to know that some of the fears which we once entertained, with good reason, are now believed to have little foundation.

It appears to me that there has been a tendency among many writers on sanitary subjects to exaggerate the dangers of contracting disease from eating meat as it is exposed for sale in the ordinary markets of the world. This tendency is to be deprecated, because it is a serious matter to arouse the suspicions of the masses in regard to a staple article of food, of which in most countries they already consume too little. A well-nourished people is a contented and healthful people, and it is the duty of sanitarians to assist in attaining this condition, rather than to make its accomplishment more difficult, by arousing groundless suspicions and fears in connection with one of the cheapest and best constituents of the diet.

As already stated, some of the reasons which were once held for accusing meat as a disseminator of disease are undoubtedly untenable with the recent advances of science; others may prove to be equally groundless. Is it not best to maintain a wise conservatism, and prove the danger before we herald it abroad? But this is not intended as an argument against inspection. On the contrary, I hold that inspection should be extended, made more thorough, and that carcasses of diseased animals, now allowed to go to the meat markets of many countries, should be condemned and destroyed. By so doing any possible danger would be removed, and the confidence of the people in their daily food would be increased.

## The Inspection of Meat with regard to the Prevention of Disease.

BY

FRANCIS VACHER, F.R.C.S., Medical Officer of Health, Birkenhead.

A truth which modern research has forced upon the attention of the medical profession is that there is no line of separation marking off the diseases of animals from those of man. One can only wonder that such an obvious truth was not clearly recognised long since. The mistake has been that the conditions of health and causes of disease in man have been studied by physicians and medical officers of health, while

the conditions of health and causes of disease on the lower animals have been studied by veterinarians. Let it be recognised that both studies are closely related branches of the same subject, and will be most profitably pursued together, and very great advances on our present knowledge may be confidently looked for. Our large towns are schools in which the laws of the health of the people are learned, and our large hospitals are schools of human pathology; but comparative pathology, though taught in the veterinary colleges, is not studied as it might be by medical men and veterinarians at every public abattoir. Unfortunately, in this country, public abattoirs are not as generally provided as they should be; still, in those we have, abundance of pathological material is from time to time presented and wasted. If the question be asked, why is this? the only answer is that we have as a nation no properly organised system of meat inspection. In a few isolated districts some special attention is given to the matter, but there is no general systematic inspection of meat with regard to the prevention of disease, and no facilities are offered for the study of diseases of animals used for food of man. Shall I say this is due to divided authority, insufficient powers, and neglect or inadequate use of powers? Doubtless it is, but the main defect is that the nation has never sufficiently realised the importance of the due inspection of meat to be used for human food.

As I have again and again pointed out, inspection is often the merest farce, split carcases, dressed and cold, being viewed and passed without the offal or any part of it being produced. Whether a carcase is fit for food is commonly decided by a nuisance inspector or market constable. When the inspection is done by a medical man, there is no guarantee that it will be more thorough. Witness Question 3479 in the Report of the Glasgow meat case heard in May 1890. (The Glasgow Local Authority v. Hugh Couper and Charles Moore). A health officer and inspector under the Contagious Diseases (Animals) Act, was asked, "How many hours in one day do you take to inspect 5,400 head?" "About three or four hours" was the answer. Private slaughter-houses are allowed almost everywhere, being often the only slaughter-houses in large urban districts, and in these efficient inspection is impossible. Inspection, however perfunctory, it may be said, is better than none. Frequently there is none, for there is little or no check on diseased oxen or sheep or swine, or even on animals found dead, being dressed in rural districts and brought into towns uninspected.

Finally, when notwithstanding the difficulties in the way of inspection, or the carelessness or incompetence of inspectors, a carcase infected with tuberculosis or anthrax is seized, it has to be taken before a magistrate, and if he refuse to condemn, it finds its way back into the meat market, and it is open to the owner to bring an action for damages against the person who seized the meat, or the authority he serves. I have had personal experience that such actions may be brought successfully.

I am aware that, owing to provisions in local Acts of Parliament and special regulations made by local authorities, the meat-supply of some districts is subjected to careful inspection, leaving little to be

desired. However, this is quite the exception, and the problem presented for solution is how to make it the rule. I have after much reflection formed definite opinions as to what is needed. The reforms I propose are far-reaching, and will not be easily effected, still nothing less will accomplish the object sought. They are as follows :—

1. *The General Provision of Public Abattoirs.*—This is the initial step. If insanitary slaughter-houses are to be abolished, better accommodation for the slaughtering of animals used for food must first be provided. I may instance the plan, &c., of the provision made in my own town in 1887. This abattoir, as experience has shown, will fairly meet the requirements of a large urban district, and I venture to think that its situation and arrangement may serve as a guide to those who contemplate the erection of such a building. It is situated on a triangular site at the outskirts of the town, bounded on the north-west by the main road along which cattle arrive from the country, on the east by the river, and on the south by a considerable extent of unoccupied land. The building is in plain style, of brick and terra-cotta, and thorough ventilation is secured. From the front are two entrances, one opening to the south avenue, and one to the main avenue. From the south avenue, access is had to three cattle lairages and to two sheep and calf lairages; there are also a house for suspected meat, a small extra lairage, and an office. Passing through any of the lairages, one enters a slaughter-house to which all lead. Beyond this, across a passage, is the cooling-house or dead-meat market, the opposite side of which opens on the main avenue. Many hanging runners extend from the slaughter-house to the cooling-house, so that carcases as soon as dressed, can be at once pushed on into the cooling-house. North of the main avenue is the superintendent's office, a store-room, lavatory, weighing-office, and boiler-house, together with the accommodation provided for the pork butchers, *i.e.*, a cooling-house, a dressing-house, two killing pens, an extra boiler house, and an extensive range of pig lairs. The main avenue communicates with the south avenue by means of the east avenue; and between the east avenue and the river is a dissecting-room, tool-house, gut-dressing room, drying-room, store-room, latrines, &c. The lairages afford accommodation at the same time for 156 head of cattle, 126 calves, 620 sheep or lambs, and the 12 lairs for swine would afford ample space for 156 full grown animals.

As under clause 169 of the Public Health Act, 1875, and allied clauses in similar Acts, "Any urban authority may, if they think fit, provide slaughter-houses," all urban districts are fully empowered. If the powers had been compulsory instead of permissive, all urban districts would long since have had public slaughter-houses. The main difficulty is as regards rural districts. Some, indeed, think that public slaughter-houses, though useful and necessary in towns, are not required in rural districts. This opinion is probably due to an exaggerated idea of what is meant by a public slaughter-house. It may be but a small place where the requirements of the district are small. The requisites are accommodation for lairing, killing and dressing, and cooling; and these may be provided in a single building having three compartments.

All that is essential is that it be quite separate from any other building; that the floors and walls (to the height of 6 feet) be impermeable, that it be well ventilated, drained, and supplied with pure water, and that it be well lighted, allowing killing and dressing always to be done with closed doors, and that the direct rays of the sun be excluded. A slaughter-house fulfilling these conditions, and suitable for a district of 10,000 inhabitants, might be erected at a cost not exceeding 6*d.* or 8*d.* per head.

What, then, is wanted? Simply compulsory powers for all sanitary authorities, enabling and requiring them to provide and maintain public abattoirs.

II. *The Closing of Private Slaughter-houses.*—From all parts of the kingdom accounts have been given of private slaughter-houses originally ill-drained or undrained, badly lighted, and unventilated, and suffered to get ruinously out of repair, and foul beyond description. The situation is often close to squalid courts and alleys, in the most densely crowded localities. The resulting effluvia are a constant nuisance to the neighbours, night is made hideous by the bellowing of beasts and the swearing of men, the frightened cattle arriving are a daily source of danger and little children grow up familiar with scenes of bloodshed. It has been proved beyond doubt that private slaughter-houses do not afford butchers ordinary facilities for cleanly slaughtering, and the efficient inspection of them or of the meat prepared in them is impracticable. There are many advantages which would result from the suppressing of private slaughter-houses and replacing them with properly constructed public ones (the removal of many standing nuisances, the checking of cruelty, &c.); but the main advantage is, that by this means only is efficient inspection of meat possible, and some security given that animals intended for the food of man will be killed and dressed under wholesome conditions.

Although, nominally, licences are granted to private slaughter-houses for one year only, many butchers believe that when once a slaughter-house has obtained a licence it is practically licensed for all time, and no matter how ruinous or filthy it may become, the local authority must renew it year by year. Though this is certainly not true, legal decisions have been given which show that a local authority cannot discontinue to licence a licensed slaughter-house without giving good grounds for so doing. However, that there can be any property in a licence, except for the period covered by the licence, will scarcely be maintained. If properly constructed public abattoirs be provided, the tenants of a large proportion of the private slaughter-houses would not ask for a renewal of their licences; and if the terms for the use of the public abattoir were very moderate (as they should be), all butchers would soon see that it was to their interest to discontinue the use of confined premises in the rear of their shops, and to let their customers know that their meat was dressed and prepared under full inspection.

III. *The Licensing and Registering of all Butchers and their Premises.*—By the closing of private slaughter-houses the local

authority would lose what little control they have over the retail trade, unless some provision were made for the licensing of butchers and their premises. It is generally admitted that the registration of dairies, cowsheds, and milkshops, and the regulations made under the Contagious Diseases (Animals) Acts, 1878 and 1886, and the Dairies, Cowsheds, and Milkshops Orders, have done much to protect milk from contamination. I propose that butchers should be dealt with in a similar manner, *i.e.*, that they be required to register and obtain licences for their trade premises, and be thus brought under the control of the sanitary authority. How this is brought about, provided it be done effectually, is not material. The Local Government Board is the fit central authority to be entrusted with the regulation of a trade, for securing cleanliness and the prevention of disease. A brief clause in a Government Act would give the Board the necessary power, and then the Board could draw up and issue byelaws for the licensing of butchers and their premises, &c. They might be to the following effect:—

1. Every local sanitary authority shall keep a register of persons carrying on in the district of the authority the trade of a butcher, and of the premises occupied by them for the purposes of their trade, and shall, from time to time, revise and correct the register; and the authority shall from time to time give public notice of a licence and registration being required, and the mode of obtaining a licence and of registering. It shall not be lawful for any person to carry on in the district of any local sanitary authority the trade of a butcher unless he be licensed and registered as such therein.

2. It shall not be lawful for any person following the trade of a butcher to occupy as a butcher's shop, or for the purposes of his trade, any premises, whether so occupied at the date of the issue of these byelaws or not, unless and until he first make provision, to the reasonable satisfaction of the local sanitary authority, for the ventilation, drainage, cleansing, and water-supply of the same, and for the protection of meat therein against infection and contamination, and until the said premises be licensed and registered by the local sanitary authority.

3. Every person following the trade of a butcher shall keep a daily journal, to be produced at all reasonable times to any officer of the local sanitary authority, in which shall be entered every purchase of meat by the butcher, and the weight thereof; the vendor's name, and a record of all sales.

4. Every local authority shall make regulations for prescribing and regulating the cleansing of butchers' shops within the authority's district, and of the fittings, vessels and implements belonging to the same; and the authority shall take steps to insure that the regulations are enforced.

Any person doing anything in contravention of the above must be liable to a penalty, and the maximum penalty would have to be specified.

IV. *The Appointment of Competent Inspectors of Meat.*—I have just been speaking of butchers' shops and their regulations. Though

these premises and their contents would of course be subject to inspection, I would have all meat inspected before it reaches the shops, and by thoroughly competent inspectors. This should be done at the public abattoir, and the selection of men to discharge such a duty is no simple task. All the qualities required in a perfect inspector of meat it is impossible to find combined in one person, and it follows that in no district should the inspection of meat be intrusted to one person, as it is often at present. Connected with every public abattoir should be a primary inspector or sub-inspector. There is no objection to the superintendent of the abattoir being appointed to this post, provided he be qualified. The best man for the place is, in my opinion, a man brought up as a butcher, who is a fair judge of meat, familiar with the appearances of healthy viscera, and not without some knowledge of the tricks of the trade. To every public abattoir should also be appointed a veterinary surgeon, who might be entitled "veterinary inspector." He would act in conjunction with the primary inspector, and advise him as to the interpretation of morbid appearances, &c. The veterinary surgeon appointed under the Contagious Diseases (Animals) Acts in any district, would be probably best suited for this office. Above these officers, and acting in all cases as the chief inspector, must be the medical officer of health. However capable the veterinary inspector and the primary inspector, the responsibility of deciding what shall pass and what shall be seized must always rest with the medical officer of health. The question to be decided on behalf of the public is not one of pathology. It is not: Is this tuberculosis generalised or localised? It is:—Is this meat fit for food of man or not? This question the medical officer of health answers after examining the carcase and viscera and hearing the veterinary inspector and primary inspector. The medical officer of health cannot put this burden on any one else's shoulders, and he should clearly understand this and qualify himself to give a right answer in all such cases. This is but one of many duties which the medical officer of health is called upon to perform, and however able, one cannot expect him necessarily to be a pathological expert and bacteriologist. Every public abattoir requires a further officer, a pathological expert, who would be consulted when necessary, and who would examine and report on specimens sent to him. The best man for this appointment will ordinarily be the professor of pathology in the nearest medical school. It may seem extraordinary that four regularly appointed officers are required for meat inspection at every public abattoir, but if the work is to be efficiently done none less will suffice. Ordinarily, only one of the four would give the whole of his time to the work. The same pathological expert might be appointed for many public abattoirs, just as the same analyst is often appointed for many districts. I pass on now to a subject kindred to the appointment of meat inspectors.

V. *The General Systematic Inspection of Animals and Meat to be used for the Food of Man.*—If killing and dressing animals to be used for human food be only allowed at public abattoirs, systematic inspection becomes a comparatively simple matter. The animals would be received at the lairages forming part of the premises at the public

abattoir, and left there for so many hours previous to slaughter. They would then come under the observation of the veterinary inspector in his daily visits, and any obvious physical signs of disease in the live animal would be noted. As I propose that master butchers should be licensed, there would no great hardship in requiring all journeymen butchers and slaughtermen employed at the public abattoir to be licensed. The sanitary authority would thus have direct control over them, and would secure their obedience to all abattoir regulations. One of the most important regulations would be the keeping of the offal of each carcase separate, and submitting it for inspection with the carcase.

Every animal brought to a public abattoir is of course booked, to insure correct returns of slaughter-house dues. If the carcase be found perfectly sound, the entry in this book should be initialled by the veterinary inspector and sub-inspector, signifying that it is so, and it may then be removed by the owner. If, on the other hand, the carcase be found diseased in any way, the entry in the book should be marked "D," the carcase and offal should be removed to the post-mortem room or other suitable locked room, and advice sent to the medical officer of health. With as little delay as possible this officer should examine the carcase and offal. If he thinks the meat may pass, he makes an entry to that effect in his book, which gives authority to the person in charge of the abattoir to have it carried back to the cooling-room. If, on the other hand, he is of opinion that the meat should be seized, he takes steps for having it submitted to a justice, and for applying for an order to destroy it. If there be insufficient evidence as to the nature or extent of the disease, the expert appointed by the sanitary authority should be consulted, and portions of the carcase and offal sent to him. The carcase should not be disfigured in any way till the justice has ordered it to be destroyed. If the hearing of an application for an order to destroy the carcase be adjourned, the carcase in the interim should be kept in the locked room at the public abattoir. If the adjournment be for several days, the carcase should be kept by the sanitary authority in a chill chamber.

The last matter I have to refer to is:—

VI. *The Appointment of competent Assessors to sit with Magistrates and assist them when necessary in the hearing of Cases relating to Diseased Meat.*—All the reforms hitherto referred to can be of little avail if, when application is made to a justice for an order to destroy a diseased carcase, the application be refused. The slaughtering of animals may be done in a suitable building under proper supervision, butchers may be licensed, capable inspectors may be appointed, and all meat prepared for food may be carefully inspected; but if the justice refus to order the destruction of the diseased meat when discovered an seized, it has to be returned to the owner, and is eventually sold to th public. The action taken in the interest of the public has cost time and money, and no one is benefited. The sanitary authority is discredited, the consumer is deluded with a sense of false security, and the butcher openly sells meat tainted with anthrax or tuberculosis. It is not the

occasional leniency of the bench in dealing with meat cases that is complained of, it is not that bias against the sanitary authority is ordinarily manifested; it is simply that lay magistrates, and even stipendiaries, are not good judges of such very technical matters as are submitted to their intelligence in "meat cases." If, when a judge is trying a case dealing with shipping matters, a nautical assessor is allowed him, is it not reasonable to ask that he be allowed a scientific assessor in trying a case when the issues depend on difficult questions of morbid pathology? Without such assistance the magistrate may be at his wits' end, trying to hold the scales of justice evenly in the midst of conflicting scientific testimony, the very language of which is foreign to him. The applicant is embarrassed with inquiries as to what would be the precise effect on the human subject after the ingestion of the meat in question, and ultimately the only way out of the difficulty (scientific witnesses being as plentiful on one side as the other) is for the magistrate to personally examine the meat and judge by the gross appearance. This is quite as likely to lead the magistrate wrong as right, for meat from a badly diseased animal may look perfectly wholesome. Indeed, if meat were condemned or passed merely on its external appearance, no elaborate system of meat-inspection would be needed. Any market constable—nay, any housewife—is able to judge whether meat be fresh or stale, whether it be young or old, whether it be in good condition or poor condition.

I submit my proposals for discussion with the assurance that they will receive thoughtful attention. If it be alleged that they are revolutionary, I will not deny it; no slight change will accomplish what is required. My suggestions are at least those of a practical public officer who knows how meat inspection should be conducted, and how it is conducted. I am no mere theorist advising others and doing nothing myself. In my district may be seen public abattoirs as complete as any I know of, and you will find but two private slaughter-houses, nor of these have I any occasion to be ashamed. We have no official butchers' register, because we are no empowered to license or register butchers, but they are under regular inspection by a capable inspector. The veterinary surgeon appointed under the Contagious Diseases (Animals) Act co-operates with the primary inspector, whose whole time is devoted to meat-inspection. All the work is systematically carried out, and is carefully supervised by myself. If the interesting pathological material from time to time met with at my sanitary authority's abattoir is not utilised as fully as it might be, this is simply due to want of time. That such material has been of immense use to me, and to those who act with me at the abattoir, in giving us an insight into animal diseases is undeniable.

## DISCUSSION.

**Professor Ostertag** (Stuttgart); speaking in German, said that at the opening of the Congress it was remarked that England was the cradle of hygiene. It was, therefore, surprising to find that meat inspection, one of the most important branches of public sanitation, had been so thoroughly neglected, notwithstanding the fact that it might so easily be enforced. He must say that he was greatly shocked to find, in a model city like London, a slaughter-house of a character that defied description. Diseased organs were lying about the yard, some of which he was told were given to pigs; the interior of the house was remarkable only for its dirt. What then must be the condition of the hundreds of private slaughter-houses distributed throughout London and England? The reasons for the absolute necessity for the adoption of a general inspection of meat were perfectly apparent. Such sources of danger as trichinæ, the larvæ of taeniæ, and echinococci on the one hand, and tuberculosis, anthrax, meat poisoning on the other, threatened the consumer of uninspected meat, and could not be guarded against by the individual. In his paper on the inspection of meat in the Transactions of the German Veterinary Council for the current year, the following points were mentioned as forming the foundation upon which the rules governing this question must be based:—

1. The erection of public slaughter-houses in all cities of more than 5,000 inhabitants.

2. Compelling the butchers to kill their animals in these slaughter-houses, and to discontinue the use of private slaughter-houses.

3. Professional direction of the slaughter-houses and the inspection of all animals both before and after the slaughter.

4. The provision of stalls for the sale of inferior but not unhealthy meat (not apt to cause disease in man), such as meat of an abnormal colour or odour, bloody meat, and that from sick animals. The provision of a cooking or sterilizing apparatus, for cooking such meat as might cause disease if consumed raw, but would be harmless when cooked (meat containing cystercerci, trichinæ, and tubercle bacilli).

5. The total destruction of those animals and parts of animals which are condemned as unfit for food.

6. The formation of a co-operative insurance society for the purpose of recompensing the owners of condemned cattle. It was best that this society should be conducted by the city or town, in order that the losses might be regularly distributed.

More attention should be paid to meat inspection, as a branch of education in the Veterinary Schools, than had been done heretofore. The veterinarian must be thoroughly grounded, both theoretically and practically, in this subject, so that he might be prepared to assume the great responsibilities resting upon the scientific meat inspector. It was perfectly evident that only the veterinarian was fitted to control the meat inspection, or to carry it out, for this work consisted in nothing more nor less than the practical application of his knowledge of the pathological anatomy of the domesticated animals. But during the period of transition, it would be necessary to employ non-professional persons who had taken a special course of instruction at some veterinary college or in a large slaughter-house. It might also be necessary to entrust the inspection in country districts to men of this class; but when the animal inspected was diseased, a regular veterinarian should be called

in consultation, before a decision was given. Until the enforcement of general laws governing meat inspection, at least the animals that were killed and were known to be diseased should be inspected before their meat went to the market.

**Dr. Geo. Fleming, C.B.** (London), said that if all the measures recommended by Mr. Vacher were carried out, the public would incur very little risk from eating flesh. The necessity for public "abattoirs" was imperative. The condition of many private slaughter-houses was positively disgusting, and reflected no credit on them as a thrifty or cleanly people. Some years ago he had occasion to examine into the condition of many of these premises in London; and he was shocked at the manner in which animals were killed and their flesh dressed for sale. Public abattoirs would have been established long ago were it not that the butchering interests could make themselves too strongly felt in the House of Commons. Public abattoirs were valuable because they were open for inspection; animals were examined as to their condition before and after slaughter; and valuable information could be gained as to the prevalence of certain diseases among food-producing animals. Gross cruelty to animals was often perpetrated in private slaughter-houses, but in public abattoirs this would be prevented, and many improvements in slaughtering, dressing, and preserving flesh could be introduced, which could not be introduced now. He insisted strongly on medical students being taught something of comparative pathology instead of comparative anatomy, in order to fit them to deal with diseases communicated from animals. For many years he had insisted on this.

**Professor Walley** (Edinburgh) was very pleased to find that Mr. Vacher practically agreed with him in his oft-reiterated statement that the inspection of meat should be carried on conjointly by the medical man and the veterinary officer: the latter being particularly useful in dealing with the characters of disease and in distinguishing between the flesh of different animals, *e.g.*, as between that of the horse and the ox. He (Prof. Walley) especially directed attention to the necessity of providing a cooling-house in all abattoirs, and of paying strict attention to cleanliness. He considered that the process of applying to a magistrate for an order to enable inspectors to confiscate meat was a roundabout process, and could be better met by the simple rule followed at Edinburgh, viz., by asking the owner of a condemned carcass to sign the confiscating book, and if he refused to do so, to prosecute him. As a matter of fact, the necessity for doing this arose only on very rare occasions. He referred particularly to the necessity which existed for making strict search for tubercular glands, and illustrated, by means of a coloured sketch, how easily such glands might be overlooked. With regard to Dr. Fleming's suggestion that veterinary surgeons should qualify themselves for the office of inspectors by taking a medical degree, he was of opinion that the degree of M.R.C.V.S. was quite a sufficient qualification. He believed that the present generation of veterinary surgeons, if taught as they should be, were better qualified to act as inspectors than were their immediate predecessors.

**Mr. Matthew A. Adams** (Maidstone) suggested that the regulations proposed by Mr. Vacher should include some provision to secure due control over slaughter in country districts: for instance, in those cases where the farmer slaughtered on the premises, and subsequently, for the purpose of sale, transported the carcasses to another—generally an

urban—district, often at a considerable distance. They were at present deprived of the means of obtaining evidence of visceral or other disease, and the history and condition of the slaughtered animals could not therefore be traced.

**Professor Brown, C.B.** (London), said that it had been the effort of the Royal Veterinary College to arrange for the study of comparative pathology at the abattoirs; but there was the difficulty of numbers. With 300 students in attendance, this would be exceedingly awkward without elaborate machinery and arrangements occupying much time daily. They could not get attendance at private abattoirs owing to the opposition of the owners, and public abattoirs were rented by private persons. There was a manifest advantage in studying morbid specimens as taken from the animal; but to duly utilise it they must advance in the study of biology. At present much biological material was wasted in the most reckless way. He was in favour of the inspection of meat as to its sufficient dryness, proper colour, and good quality. At the same time, as Professor Walley had shown, it was quite possible for an animal with tuberculous bones to be sold as meat, without the slightest suspicion of its being diseased in any part of its structure. Undoubtedly the seizure of carcasses involved hardship. Not long ago the butchers had forcibly complained to the Board of Agriculture that they bought animals in the open market at a large price and to all appearance perfectly healthy, but that when, after slaughter, they were found tuberculous, the carcasses were seized, and they had no remedy whatever. Here, again, the question of compensation cropped up, and there was an almost insurmountable difficulty in obtaining what was necessary. Many years ago this matter was dealt with by an Act entitled "The Buildings Improvement Act," but it proved a remarkable instance of failure of legislation. Something like 34 years were to elapse from the passing of the Act until its coming into force. No one took any notice of the Act until this interval was about to expire, when that portion of it referring to slaughter-houses was actively opposed on the ground that it would have the effect of shutting up every slaughter-house in the country. The result was that that portion of the Act was repealed by a short Bill. If, however, the Legislature could be induced to insist upon animals being killed in public abattoirs, the benefit to public science would be immense. Certainly, from the point of view of the Veterinary Department, it would be an immense advantage to be able to proceed to the actual place where animals were killed, and obtain indisputable reports as to the absence or existence of disease. Butchers were very much in favour of private slaughter-houses. In some cases animals were killed in the shop itself, only a curtain separating the place of slaughter from the public view. With reference to registration and licensing, the former would certainly present no difficulty; but there was a considerable difficulty as to the licensing of anything. But even registration alone would be a step in the right direction, because it would be extremely convenient to have access to a list at the central authority's office which would at once put them in possession of the address of any butcher in the district. At present there was no such list, and they were commonly put to considerable difficulty in order to obtain information. He thought that at least some of the provisions suggested by Mr. Vacher would before long be carried into effect.

**Professor McFadyean** (Edinburgh) said that he did not quite agree with the scheme formulated for the supervision of public slaughter-

houses and the general inspection of meat. In point of simplicity and efficiency he much preferred the system of inspection in force in most of the large Continental abattoirs, such as those of Leipzig and Munich.

**The President** said that the Section would now adjourn to the rooms of the Royal Society to take part in the debate then proceeding in Section II., on Tuberculosis.

[For the Discussion on Tuberculosis, see *Volume II.*, p. 204. The two papers originally contributed under the following titles to SECTION III., have been transferred to SECTION II., and will be found in connection with others dealing with the subject of Tuberculosis on pp. 193–7, Volume II.]

## Le Danger supposé de la Consummation du Lait et de la Viande sains en apparance mais provenant d'Animaux atteints de la Tuberculose.

PAR

le Professeur BANG, Copenhagen.

(*Vide Volume II.*, p. 193.)

## On the Transmission of Tuberculosis from Animals to Man by means of Flesh and Milk derived from tuberculous Animals.

BY

Professor MCFADYEAN, M.B., B.Sc., F.R.S.E., Edinburgh, and
G. SIMS WOODHEAD, M.D., F.R.S.E., London.

(*Vide Volume II.*, p. 197.)

## Friday, 14th August, 1891.

The PRESIDENT, Sir NIGEL KINGSCOTE, K.C.B., in the Chair.

VICE PRESIDENTS of the day:
D. E. SALMON, D.V.M., Washington, U.S.A.
Professor G. T. BROWN, C.B., Director of the Veterinary Department of the Board of Agriculture, London.

## Maladies Infectieuses des Animaux qui peuvent être transmises à l'Homme, et réciproquement.

PAR

le Professeur E. PERRONCITO, Turin.

Le *Proteus virulentissimus* est un microbe qui cause une maladie très analogue au charbon et à la septicémie dans les bovidés, dans les chevaux, dans les brebis, dans les porcs, et qui peut se transmettre à l'homme. Il est aérobe et anaérobe, et croît sur la gelatine, dans le bouillon, l'agar, les pommes de terre, etc. J'ai décrit ce micro-organisme dans l'année 1889*, et j'ai vu alors et ensuite qu'il peut donner dans l'homme des formes très analogues à la pustule maligne. Il se connaît parcequ'il se présent sous forme de l'élément: gros 1—2 $\mu$, long 2—3 $\mu$, avec alone ou capsule autour qui ne se colore pas; sur la surface des cultures en gelatine on trouve aussi des filaments minces, homogènes et pâles, deux à deux, ou en chenilles.

Je vous présent une préparation microscopique qui fait voir sur la muqueuse de l'estomac d'un lapin une grande quantité de *Bacillus anthracis.* C'est le Dr. Brusaferro qui dans un lapin inoculé par le charbon a trouvé sur la muqueuse de l'estomac des ecchymoses auxquelles correspondaient de grandes accumulations du *Bacillus anthracis.*

Une autre préparation vous démontre comme les abassîns sont sujets au *Tœnia mediocanellata.* Dans un jeune enfant j'en ai recueilli cinq exemplaires.

Dans la quatrième préparation vous verrez le *Trichophyton tonsurans* que j'ai décrit dans le mouton. Il a causé des plaques de *l'herpès tonsurans.*

La cinquième préparation se réfère à l'actinomycès que j'ai décrit dans 1874 comme semblable à une végetation cryptogamique. Une observation très intéressante d'actinomycès, je l'ai faite avec M. le

* E. Perroncito. Un proteo virulentissimo et la proteosi nel bestiame. R. Accademia Medica di Torino 10 Maggio 1889. Le malattie dominanti nel bestiame della Sardegna. R. Accademia d'Agricoltura di Torino 1889.

Professeur Reymond, dans la peau d'une femme (dans le 1881), et une autre, pas moins intéressante, je l'ai faite à la peau et dans le tissu sous-cutané d'un cheval, dans lequel se développait l'actinomycose, ensuite à une blessure qu'il a reçu à une cuisse. Le cheval après sa blessure a été mis dans une étable de bêtes bovines, et en cette manière peut-être s'est inoculé accidentalement l'actinomyces du bœuf.

Je vous présent le résumé de mes expériences faites sur l'utilisation des viandes des animaux tuberculeux, qui démontre comme les viandes constituent peut-être le moins fréquent des moyens de transmission de la maladie à l'homme.

Dans ces dernières années, j'ai voulu faire des nouvelles expériences sur l'utilisation des viandes des animaux tuberculeux. La viande était tirée de l'abattoir de la ville de Turin, et appartenait à bovidés tuberculeux à différents degrés. De ces viandes, j'en ai fait manger à des jeunes cochons (porcs) et plus que 14 en ont consumées par 1, 2, 3, 4 mois et plus, sans qu'ils eussent presenté ensuite les symptômes ou les lésions de la maladie transmise.

De la même viande j'ai préparé du suc pur ; on y mêla un très peu d'eau. Avec ce suc j'ai inoculé sous la peau ou dans la cavité péritonéale plus que 100 lapins et 100 cobayes. Les animaux ainsi opérés ont été conservés un mois et demi, deux mois, trois mois, et davantage, encore sans qu'aucun eut présenté les symptômes, ou à l'autopsie, les lésions de la tuberculose.

A deux vaches, préalablement rendues réfractaires au charbon avec la vaccination charbonneuse, j'ai inoculé du même suc de viande sous la peau en arrière de l'épaule. Les deux vaches ont été tenues vivantes plus que cinq mois, sans qu'elles eussent presenté les lésions de la tuberculose.

A quatre porcs de six mois, j'ai fait manger plus que quatre mois de la viande de bœufs et de vaches tuberculoses, et à deux autres porcs j'ai fait manger en outre plusieurs fois une quantité notable de nodules tuberculeux des bêtes bovines, sans obtenir la transmission de la maladie.

Ces expériences auront peut-être une influence pour ne pas attribuer une importance exagérée à l'action de la viande pour la diffusion de la tuberculose dans l'homme.

Dans les tubercules des bovidés, avec le Dr. Brusaferro de Turin, j'ai constaté plusieurs fois la difficulté qu'il y a à trouver des bacilles de Koch, et même quand on en trouve aucun, la matière est aussi contagieuse ; ce qui démontre comme les cellules géantes et les autres cellules des tubercules agissent d'une action phagocitique plus ou moins forte jusqu'à détruire le virus représenté par les bacilles. Mais il y reste toujours quelques-uns modifiés ou non, capables de transmettre la tuberculose après l'inoculation.

### *Sur la diffusion des Cercomonas intestinalis de l'Homme et des Animaux.*

Tout ce qui concerne la diffusion des espèces des protozoaires qui habitent en parasites l'intestin de l'homme et des animaux a une très grande importance pour expliquer l'étiologie de quelques maladies ou

manifestations morbides produites par leur pénétration dans l'organisme, par leur multiplication et leur genre de vie.

Dans le 1887, j'ai commencé à étudier le *Megastoma entericum* (Grassi) ou *Lamblia intestinalis* (Blanchard) dans le duodénum de l'homme et du rat.* Dans l'homme il produit quelquefois des troubles assez considérables, que j'ai indiqués dans ma note communiquée à notre Académie de Médecine. Quelquefois il se trouve seul en grand nombre, autrefois il se trouve avec *l'Anchylostoma duodenalis* ou avec d'autres espèces helminthologiques. Ce protozoaire vit et se multiplie dans le duodénum de l'homme et du rat (*Mus musculus*, *Mus decumanus.*) Pour se propager, il doit parcourir tout l'intestin et spécialement dans le colon il sépare autour de lui une membrane chitinoïde ou kyste qui constitue l'inkystement ou encapsulément du megastome. Dans cette condition mêlée aux substances de rejection, il vient éliminé à l'état de vie durable comme de spore, et se voit à l'examin des féces sous la forme de corpuscules translucides ovales, du diamètre de 10, 12, 17 $\mu$ pourvu de membrane externe qui laisse voir un contenu cellulaire distinct, avec les flagelles du protozoaire même.

Que cette forme du megastome constitue le moyen de propagation de l'espèce parasitaire, j'ai pu le démontrer expérimentalement sur les rats (*Mus musculus* blanc.) Dans 1887, j'ai saisi quatre rats (Möuse) et j'ai fait manger à deux des substances qui contenaient en grandes quantités les megastomes enkystés; les deux autres, je les ai tenus comme animaux de control. En peu de jours les deux rats qui ont mangé les dîtes substances se sont présentés avec les mégastomes, au contraire les deux autres n'ont rien présenté.

Dans ces derniers mois, j'ai pu répéter les expériences sur d'autres animaux, et j'ai etabli qu'en six jours les mégastomes enkystés, mangés par les rats peuvent déjà se propager par la même forme d'évolution.

La même manière de développement se trouve dans l'homme. Les individus de notre espèce prennent certainement les mégastomes parmi les rats, qui vont à déposer ses excréments sur le pain et sur d'autres aliments. La conséquence hygiènique est très facile à déduire.

J'ai pu constater que les autres espèces de cercomonas de l'homme et des animaux se propagent en prenant la même forme d'encapsulément, comme j'ai demontré dans le 1888.†

*Sur l'enkystement des larves mûres de l'ankylostoma duodenalis.*

Comme j'ai decrit dans 1880,‡ si les œufs de *l'ankylostoma* éliminées par les individus affectés d'anémie ou pas encore anémiques, trouvent des conditions favorables, elles éclosent, les embryons parcourent la première période de vie de rhabditis libre, ensuite se transforment, la

---

* E. Perroncito: L'incapsulamento del *Megastoma intestinalis.* R. Accademia di Medicina di Torino, seduta di Maggio 1887.

† E. Perroncito. Sur la diffusion des cercomonas intestinaux, R. Académie de Médecine de Turin. Séance du 24 Février, 1888. Archives italiennes de Biologie, fascicule 11.

‡ E. Perroncito: L'anémie des mineurs au point de vue parasitologique. Archives Italiennes de Biologie. Tome II. fascicule 111.

peau sépare une membrane ou kyste qui se dispose, et qui répéte complêtement la forme du ver parfait, seulement c'est mince, et souvent très vorace; la larve perfectionne les organes internes et reste ainsi dans sa Kyste à attendre d'entrer dans le corps de l'homme, ou de quelque animal, dans lequel elle puisse parcourir les autres phases de vie pour arriver à l'état de ver parfait.

La kyste subit plus ou moins vîte la calcification, comme vous pourrez observer dans les préparations que j'ai l'honneur de soumettre à vos observations.

Dans les différentes conditions que les larves mûres se trouvent, l'espèce parasitaire peut se disséminer par l'eau, par la poudre des rues etc., comme j'ai démontré en étudiant l'anémie des mineurs du St. Gothard.*

## Actinomycosis.†

BY

Professor Crookshank, M.B., King's College, London.

I was invited by the Council of this Section to open a discussion on "Diseases communicable from Animals to Man and *vice versâ*," omitting any diseases which might be selected as separate subjects for discussion. Under these circumstances I thought it would be more satisfactory to deal exclusively with a disease common to man and cattle which has interested me for some years more than any other. I refer to human and bovine actinomycosis.

*Not a new disease.*—Actinomycosis has been frequently referred to as a new disease. Even so recently as in December, 1889, Professor Byron, Director of the Bacteriological Laboratory of New York, in describing a case in man that came under his observation, spoke of it as a new infectious disease. It is not, however, thus correctly described, for the various manifestations of it have been met with for more than half a century, though misunderstood and misnamed.

From contemporary literature there can be no doubt that actinomycosis was very prevalent in Scotland in 1827-1839, and veterinary surgeons have long been familiar with certain morbid conditions in cattle known as wens, clyers or crewels, sitfasts, scrofulous, tubercular or strumous abscesses, polypus or lymphoma or simply tumour of the throat, cancer of the tongue, scirrhous tongue, indurated tongue, ulcerated tongue,

* E. Perroncito: Osservazioni elmintologiche relative all' anemia sooltasi epidemia vegli operai del St. Gottardo, R. Accademia dei Lincei, seduta del 2 Maggio, 1880.

L'anemia dei contadioni fornaciai e minatori in rapporto coll'attuale epidemia vegli operai del Gottardo, 1880. Torino R. Accademia di agricoltura, Vol. XXIII.

† This paper was illustrated by specimens from animals affected with actinomycosis.

cancer of the jaw, bone-tubercle, osteo-sarcoma, fibro-plastic degeneration, spina-ventosa and carcinoma. But it is only comparatively recently that owing principally to the work of Italian and German pathologists, it has been found that these terms have been misapplied to the new growths associated with a highly characteristic vegetable micro-parasite, the ray-fungus or *actinomyces.*

*Prevalence in England.*—Six years ago, after studying actinomycosis in the pathological laboratory of Professor Johne of Dresden—well known for his researches on this subject—I began to collect on my return to England all the information and material I could obtain of both human and bovine cases in this country. I found that the disease was not generally recognised as a common affection in cattle, in spite of the interest excited by the work of Dr. Fleming, to whom is due the great credit of first recognising a case in this country and of bringing the work of the continential investigators prominently before the veterinary and medical professions. In 1887 I investigated a disease prevailing in Norfolk, and in the following year outbreaks which occurred in Essex, Hertfordshire, Cambridgeshire and Middlesex. In the Norfolk outbreak I found on one farm 8 per cent. of the beasts affected with the so-called "wens" or "sitfasts" which I proved by microscopical examination to be cases of actinomycosis. These growths have been regarded from casual inspection as the result of strumous or scrofulous inflammation, but in all the specimens of wens which I have received from this country and the colonies I have been able to demonstrate the presence of the ray-fungus.

A case of pulmonary actinomycosis with grape-like growths on the pleura convinced me that wens were not the only form of this disease which had been lost sight of under the designation of tuberculosis. In the morbid specimens (handed round) to which I would draw your attention you will see appearances which have been invariably attributed in this country to tuberculosis, and will continue so to be, if we rely on a hasty examination in the slaughter-house.

*Its manifestations.*—I shall proceed to refer to the various manifestations of this disease, and to illustrate my remarks by morbid specimens from my own collection and by photographs:

Taking first of all the *Digestive system*, we find the disease attacking:—

(*a.*) *The lips, gums, buccal mucous membrane and palate*, and appearing as nodules, wart-like growths or ulcers. The nodules and ulceration of the palate are well shown in a specimen sent to me for examination under suspicion of being the result of severe foot and mouth disease.

(*b.*) *The upper and lower jaw*, where it probably originates in carious teeth, thence extending and invading the neighbouring cavities and sinuses, destroying the tissues with which it comes in contact, and expanding the bones into thin plates or reducing them to the appearance of pumice-stone.

(*c.*) *The tongue*, where we see it most commonly in the form of nodules or wart-like patches under the mucous membrane with a special

tendency to ulcerate from the irritation of the teeth. These nodules may extend into the deep muscles, and often collect in rows more or less parallel to the superficial muscular fibres. Complete transverse sections of the tongue double-stained readily show this arrangement even to the naked eye (sections exhibited). Induration of the tongue results from secondary interstitial glossitis. I have seen, in one case only, a tumour embedded in the substance of the tongue about the size of a small tangerine orange and more or less isolated from any surrounding growth.

(*d.*) *The pharynx*, where the polypoid growths may be illustrated by a specimen from a most interesting case kept under observation for some time at the Royal Veterinary College. The animal died suddenly of asphyxia and at the post-mortem a tumour the size of a cricket ball was found occluding the larynx.

Taking next the *Respiratory system* we may meet with the disease in :—

(*a.*) *The nasal cavities*, originating primarily there or resulting from extension of a growth from the lips or from the pharynx, as in the specimen before you.

(*b.*) *The larynx and trachea*, generally in the form of polypoid growths, sessile or pedunculated, which arise primarily or occur secondarily by extension from the tissues in the neighbourhood.

(*c.*) *The lungs*, where the differentiation of the disease is most important, as the neoplasms in the lungs especially in the early stages and the nodular growths of the pleura are constantly mistaken for tuberculosis.

The disease is very rarely found in connection with the *Nervous system*, but probably does not so rarely attack the *Reproductive system ;* although so far as I am aware only two cases have been described, one by myself and one by Professor McFadyean.

Fifthly, in the *skin and subcutaneous tissues* we find a favourite seat of this disease, producing the so-called wens or clyers so commonly seen in the fen country. A wen is first recognised as a small tumour the size of a marble or walnut, which increases in size sometimes with great rapidity and breaks down and discharges its muco-purulent contents through the inflamed and ulcerated skin ; or it may go on increasing and form a large compact growth the size of a child's head. These growths when excised, hardened and cut, have a characteristic honey-combed appearance produced by the interlacing bands of fibrous tissue which form a spongy structure from the interstices of which the fungus-tufts and thick yellowish pus have for the most part dropped out. These appearances are seen in the morbid specimens, and in complete sections of the tumours (sections exhibited). The growths as seen in the living animal I will illustrate upon the screen (lantern slides exhibited).

*Prevalence in Australia.*—In a report to the Legislative Assembly of New South Wales, Mr. Willous, Government Veterinarian, gave an account of an investigation made in conjunction with Mr. Park into the nature of a prevailing disease of the cattle in Tasmania. The disease occurred principally in the form of tumours of the upper and lower jaw, and from the detailed description I have no doubt whatever that they were cases of actinomycosis and wrongly attributed to "scrofulous

inflammation." At the Australasian Stock Conference, held in Melbourne 1889, my report on human and bovine actinomycosis, published in the Annual Report of the Agricultural Department, 1888, was brought to the notice of the Board by Mr. Buchanan, Mr. Stanley, and Mr. Park, and from the coloured plates and photographs in the Report the nature of the disease known in Australia as cancer and lumpy jaw was at once recognised. Mr. Park moved for an enquiry, and while studying in my laboratory, has informed me that this disease is very prevalent, but not yet recognised by the colonial veterinary surgeons as distinct from tuberculosis. Mr. Park brought over a tumour which he submitted to me for examination and report, and it proved to be an exceptionally interesting case of actinomycosis which I have referred to in a communication to the Bacteriological Section.*

Dr. MacLaurin, President of the Board of Health, who was also studying in my laboratory, on his return to Sydney demonstrated by microscopical examination the true nature of the cases mistaken for tuberculosis. In his report to the Legislative Assembly of New South Wales, June 1890, Dr. MacLaurin states that the existence of the disease had not been recognised in Australia until he demonstrated the presence of the ray-fungus in a specimen belonging to Mr. Stanley.

*Prevalence in the United States.*—Reports of the prevalence of actinomycosis in the United States have reached me through the courtesy of the Board of Live Stock Commissioners for the State of Illinois. In their Report for 1890, several interesting communications have been published. Mr. Casewell, State Veterinarian, investigated an outbreak of the disease, known in America as "Lumpy jaw," on a farm in Yates City, where there were 80 head of cattle, and 16 were found to be suffering from actinomycosis. And writing from Chicago, Illinois, Mr. Casewell reported that the disease was prevalent in nearly every county in that State, and that in his opinion it was spreading rapidly, and was "dangerously contagious." In December, 1889, Messrs. Casewell, Scott, Williams and Page, reported to the Board that they had examined certain beasts quarantined in the City of Peoria, 12 bullocks in charge of the Northern Distillery Co., 33 in charge of the G. W. Distillery Co., and 39 in charge of the Monarch Distillery Co., and that all of them were affected with actinomycosis. In all, 109 animals were slaughtered and condemned as unfit for food. Mr. Baker, reporting on 4 steers and one cow, in the Union Stock Yards in Chicago, found them suffering from actinomycosis in the form of large tumours in the head, neck, and throat, and small tumours the size of a large pea in the intestines. The meat was condemned as unfit for food.

As the result of these reports the Board enforces the destruction of all animals affected with actinomycosis and prohibits the use of the carcasses of such animals for human consumption.

At a Conference of the Inter-State Live Stock Sanitary Association, the following resolution was carried: "That it is the sense of this

* On the Morphology of Actinomycosis. Transactions of the Seventh International Congress of Hygiene, Vol. II., page 105.

"Conference that animals affected with this disease should be destroyed, "and that the carcasses thereof should not be used for human food."

*The disease in man.*—As in cattle, so also in man, cases of actinomycosis have been met with, but not recognised as such. By the researches of Israel and Ponfick in Germany, and the recognition of a case in man by Acland in this country, a new field of enquiry was opened up. It was not a new disease in man, but it had not been differentiated from the diseases which it simulates.

The evidence on this point is absolutely conclusive, and it affords a striking example of the value of accurate drawings for illustrating researches on pathological subjects. In 1848, M. Louis met with a pulmonary affection supposed to be cancerous. M. Lebert discovered in the pus, and, though unable to explain them, figured the tufts of club-shaped elements with which we are now so familiar. Nearly 200 cases have since been described in the human subject, and about 5 per cent. have occurred in this country.

*Points for discussion.*—I would direct your attention to the following points as especially worthy of discussion. I referred to the manifestations of the disease liable to be attributed to tuberculosis, and would again draw attention to the morbid specimens and photographs. To what extent is this an explanation of the reports of a very high percentage of cases of tuberculosis in cattle, both in this country and in the colonies?

With regard to the disease being dangerously contagious, I am of opinion that the disease is very rarely, if ever, the result of direct infection from cow to cow, or from cow to man. Two cases have been recorded in support of the theory of infection of man from the cow. Stelzner described a case in a man who had the care of animals, some of them having suppurating glands, and Hacker met with the disease in the tongue of a man who had charge of cows, one of them having had a tumour of the jaw. On the other hand, Moosbrugger found that out of 75 cases, 54 were in men, and 21 in women and children. In 11 the occupation was not stated, in 33 their occupation did not bring them into contact with cattle. They were, for example, millers, glaziers, tailors, shop people and students; 10 cases occurred among farmers, peasants and farm labourers, and in one case only was there contact with diseased animals. Out of the 21 women and children, only four were peasants, and none had been in contact with diseased cattle.

I have so far found no evidence in this country to support the theory of direct infection from the cow, but I have succeeded in transplanting the disease from man to a calf.

There is reason for supposing that man and animals derive the disease from a source in common, and there is a strong suspicion attached to cereals. In my opinion the Royal Commission now investigating tuberculosis should include the subject of actinomycosis, with special reference to the question of the possible transmission of the disease to man, and the action to be taken with regard to the flesh of the diseased animals, and I trust that evidence will be forthcoming on these points from the members attending the meeting of this section.

# Trois Cas d'Actinomycose chez l'Homme.*

PAR

les Drs. Doyen et Roussel, de Reims.

Nous avons eu l'occasion d'observer chez l'homme, dans ces derniers mois, trois cas d'actinomycose. Cette affection est très rare en France. Si nous exceptons le cas de Lebert (1857), la première observation, due à Nocard et Lucet, date de 1888. Il y avait cependant dix ans que les mémoires d'Israël (3 observations), de Ponfick (1 observation) et les nombreux cas publiés en Allemagne attiraient sur cette nouvelle maladie l'attention des bactériologistes français. Depuis 1882 nous n'avons négligé l'étude microscopique d'aucun cas de suppuration et de septicémie, et c'est seulement en Février 1891, à quelques jours d'intervalle, qu'il nous a été donné d'observer nos deux premiers cas. Le premier malade nous consultait pour un abcès indolent de la joue. Les parois étaient indurées ; la cavité, du volume d'une noisette, contenait, dans une sérosité sanguinolente, une cinquantaine de grains d'actinomycès. La poche fut extirpée en totalité. Le second cas se rapporte à un phlegmon profond de la base de la langue, avec induration ligneuse de toute la région sus-hyoïdienne. (Académie de Médecine et Congrès de Chirurgie ; 29 Mars et 1er Avril 1891.) La guérison fut obtenue par l'incision large, suivie du curettage et du tamponnement antiseptique.

Nous commençâmes immédiatement les recherches bactériologiques. Les cultures du liquide sanguinolent du premier cas et du pus phlegmoneux du second restèrent stériles ou donnèrent, pour cas derniers, quelques colonies de *Staphylococcus cereus albus*. Nous avons, au bout de quelque temps transplanté sur d'autres tubes les grains demeurés vierges de toute contamination. Un troisième cas nous préoccupait : celui d'une périty phlite suppurée à marche chronique, dont le pus était resté stérile, lors de l'incision (fin Novembre 1890) lorsqu'un de nous (Juillet 1891) put recueillir à l'orifice de la fistule deux petits grains jaunâtres d'actinomycès. Un phlegmon profond se développa, par propagation du côté gauche, et nous donna à l'incision près de 1 litre de pus phlegmoneux et plus de 1,000 grains caractéristiques, le pus demeura complètement stérile en bactéries pyogènes. La culture des actinomycès est poursuivie en ce moment dans mon laboratoire.

Il nous a paru intéressant de comparer nos préparations d'actinomycose de l'homme à celles que nous possédions de l'actinomycose du bœuf. Nous avions trouvé en effet au premier examen, des filaments ramifiés, à disposition radiée, se colorant par la méthode de Babès, mais il nous était impossible de mettre en évidence les figures si caractéristiques de l'actinomycose du bœuf. Notre second cas nous donna des préparations analogues. Le troisième offre peut-être à côté des

* This paper was illustrated by a demonstration of numerous fine lantern slides.

actinomycès, un autre mycélium qui sera ultérieurement déterminé. Les méthodes de double coloration ne nous ont pas permis de voir les filaments ramifiés se terminer dans des crosses comme on l'observe si nettement sur cette photographie (*shown on screen*) d'une belle préparation de M. le Professeur Cornil, mais provenant de l'actinomycose du bœuf.

Beaucoup de filaments présentent des parties incolores, constituées par une sorte de gaine où se montrent des grains ovalaires qui semblent être des spores—quoique tous se terminent par un petit bouton arrondi. La dissociation des parois de l'abcès du premier cas, et quelques grains des deux autres nous ont enfin donné des aspects tout particuliers, l'actinomycès s'y montrant sous forme de courts batonnets incurvés comme des spirochætes, mais présentant le plus souvent plusieurs ramifications dichotomiques; ce sont des formes jeunes du champignon.

Les coupes des parois du foyer extirpé ne nous ont donné aucune des figures rayonnées caractéristiques chez le bœuf, mais seulement des éléments isolés, assez analogues aux crosses par leur forme et leur réaction vis-à-vis des couleurs d'aniline, et quelques cellules remplies de courts batonnets ramifiés.

Nos cultures et nos inoculations sont encore trop récentes pour que nous puissions en donner tous les caractères. Les grains s'implantent lentement à la surface des tubes d'agar et de sérum. Les tubes de bouillon ne sont pas troublés, et le champignon y prend la forme bien connue de l'espèce "Cladothrix" celle d'une petite sphère blanchâtre revêtue d'un fin chevelu. L'examen des cultures révèle de nombreux renflements à l'extrémité des filaments mycéliens.

On a signalé partout la rareté des crosses dans les cas d'actinomycose de l'homme. Les tumeurs d'apparence sarcomateuse, et que l'on rencontre chez le bœuf, ont sur les coupes un aspect tout autre que les parois de l'abcès de notre premier cas.

Les caractères différentiels qui nous semblent dès aujourd'hui se manifester dans les cultures sur les milieux solides, les tendances du champignon que nous avons observé à se montrer un organisme pyogène, capable de déterminer par lui-même l'inflammation, la suppuration et la fièvre, nous portent à émettre l'hypothèse qu'un certain nombre des cas décrits comme actinomycose chez l'homme different essentiellement de l'actinomycose du bœuf.

La réaction variable des tissus de diverses espèces animales (tumeurs inflammatoires ou suppuration) ne pouvant suffire pour différencier l'une de l'autre deux bactéries analogues, nous devons attendre le résultat ultérieur de nos cultures et de nos inoculations pour conclure s'il existe ou non, parmi les cladothrix, plusieurs espèces pathogenes voisines de l'actinomycès. Nous croyons devoir insister sur ce fait qu'en France l'actinomycose est rare chez les animaux. Chez l'homme, depuis le cas de Nocard, il n'en existe que quatre observations précises, une toute récente, et encore inédite étant due à notre ami le Dr. Netter (pleurésie purulente). L'observation de Darier se rapporte à une malade d'origine allemande. L'actinomycose comptant, par ses manifestations viscérales au nombre des maladies infectieuses les plus

grandes, il y a lieu de se préoccuper de l'extension que pourrait prendre, en France, cette nouvelle affection.

Au point de vue de l'étiologie, les trois cas que nous avons observés proviennent des environs de Reims, les trois malades habitent ou ont habité la campagne, et l'un d'eux, notamment, avait l'habitude de mâchonner sans cesse des grains d'orge et de blé.

## DISCUSSION.

**Prof. Ponfick** (Breslau) drew attention to the fact that the disease of actinomycosis was usually transmitted from fodder, especially from straw. He cited an instance of a boy who was playing with a barley awn. In drawing it rapidly from his mouth a small particle became lodged in the pharynx, and passed down into the lungs. At the end of eight weeks an abscess appeared under the scapula. The abscess was opened, and out came the fragment of the awn of barley which had been swallowed. Then actinomycosis made its appearance in connexion with the ribs and lungs, which, however, presented very much the appearance of tuberculosis. From this case Prof. Ponfick argued that the cause of the disease in man was practically the same as in animals. It was probably through external materials finding their way into the alimentary canal, just as in cattle. In fact, there was not a direct transmission from animals to man, but a common source of the disease obnoxious to both.

**M. Nocard** (Alfort) dit:—Je ne traiterai pas la question scientifique. M. le Professeur Crookshank l'a fait d'une façon magistrale. Je voudrais envisager le côté hygiénique et sanitaire de la question. Lorsqu'on étudie la distribution géographique de l'actinomycose, on est frappé de son irrégularité; tel pays, telle province, tel district en est littéralement infecté, tel autre en est absolument indemne. La Bavière, l'Ecosse, l'Italie, certains Etats de l'Amérique du Nord ont beaucoup d'actinomycose ; à l'Ecole Vétérinaire d'Utrecht l'hôpital renferme toujours plusieurs sujets atteints de la maladie; quand j'ai besoin de pièces fraîches pour mes démonstrations pratiques, j'écris à mon collègue Thomassen et dans les huit jours, je reçois une " langue de bois ", comme on dit en Hollande. En France, l'actinomycose est très rare; on ne l'y voit guère que sous la forme d'ostéo-sarcôme de la mâchoire. Voilà tantôt dix ans je la recherche avec soin dans les autres organes, examinant tous les pus, toutes les tumeurs d'aspect sarcomateux ou tuberculeux que je rencontre dans mon service ou que m'envoient mes anciens élèves; et je n'ai vu jusqu'ici que trois cas d'actinomycose pulmonaire (un cas de Moulé, un cas de Leclerc, un cas de Greffier), et un seul cas d'actinomycose du rein chez un porc (Nocard et Moulé). Même sous la forme de tumeur de la mâchoire, la maladie reste toujours, chez nous, à l'état isolé; jamais on ne la voit se transmettre aux voisins du malade, et cependant, ce malade reste toujours très longtemps, souvent plusieurs années, dans l'étable, au contact intime de ses voisins. Tant que les progrès de la tumeur n'ont pas rendu impossible la mastication des aliments par l'ebranlement ou la chûte des molaires, le malade reste vigoureux; il mange, il engraisse, il travaille, il produit du lait comme ses voisins. Souvent on voit la tumeur se ramollir en un point, s'ulcérer et donner écoulement du pus

très riche en actinomycès; l'orifice ne se cicatrise qu'avec difficulté; il demeure à l'état de fistule, donnant incessament issue au champignon pathogène; et cependant, je le répète, jamais en France, on ne voit les voisins du malade devenir malades à leur tour. Comment donc se fait l'infection ?

Il ne paraît pas douteux qu'elle provienne des aliments. Plusieurs observations précises tendent à établir que les germes du parasite sont introduits avec les graminées; le Professeur Piana notamment a cité un fait dans lequel il a trouvé au centre de la néoplasie linguale une *Carbule* d'orge, d'où l'actinomycès avait manifestament rayonné dans toute l'épaisseur de l'organe.

Dans ces conditions, on conçoit que, là où les pâturages sont infectés des germes du parasite, beaucoup des animaux qui y pâturent, puissent s'infecter à leur tour en y prenant leurs aliments; c'est ainsi que le Professeur Hueppe me disait tout à l'heure connaître une grande exploitation où 60 bœufs ou vaches étaient en même temps atteints de la maladie sous des formes variées. On conçoit aussi que, dans ces districts, l'homme puisse également s'infecter par l'usage d'aliments herbacés, qui n'ont pas été soumis à la cuisson. Il n'est pas besoin d'invoquer la contagion de l'animal à l'homme ou réciproquement.

C'est pourquoi, je ne crois pas utile de prendre à l'égard des bovidés malades des mesures de police sanitaire spéciales; aux Etats Unis, par exemple, il semble qu'une véritable panique se soit emparée des esprits; on traque les bœufs atteints d'actinomycose; on les abat, on les détruit sans permettre l'utilisation de la viande ou des débris. Rien ne motive une pareille exagération; non seulement elle est très couteuse, mais encore elle est inutile. Les pâturages infectés n'en continuent pas moins à donner des fourrages chargés des germes du parasite. A mon avis les efforts des hygiénistes doivent tendre à mieux connaître la biologie de l'actinomycés en dehors de l'organisme; peut-être pourra-t-on alors en déduire une prophylaxie rationelle et efficace.

**M. Serge Ivanof** (Moscow), dit; L'actinomycose est une maladie très répandue chez les bovidés. Comme chef de service de l'inspection des viandes à Moscou, j'ai vu plus, de 2,000 animaux actinomycotiques pendant deux années de mon service. Voilà ce que je puis conclure de ma pratique: l'actinomycose est une maladie bénigne et tout-à-fait localisée dans le corps de bovidés. Les cas généralisés ne se rencontrent pas souvent; au contraire ce sont des lésions peu étendues et récentes, que j'observais dans la majorité des cas. Pendant la première année de mon service, mes collègues et moi nous employons seulement l'inspection microscopique de tumeurs et d'abcès suspects, et nous n'avons vu que moins de deux cents cas de cette maladie; mais ensuite, avec l'emploi de microscope, nous avons observé que l'actinomycose se rencontre souvent sous forme de petites tumeurs dans la peau du cou, qui ne sont pas plus grandes que l'œuf de poule, ou de petites tumeurs sur la muqueuse de la bouche, qui ne dépassent pas le volume d'un pois. Je pense que dans la statistique des abattoirs de plusieurs grandes villes, où l'on n'observe pas beaucoup d'actinomycose chez les animaux, c'est probablement parce qu'on néglige de rechercher ces lésions du début, de beaucoup les plus fréquentes.

**Dr. D. E. Salmon** (Washington, U.S.A.), remarked that the statements in regard to the prevalence and contagiousness of actinomycosis in the United States had been greatly exaggerated, because of the financial interests involved and the rivalry between State and City

inspectors of animals and meats. Conclusions drawn from such statements were liable to be very far from the truth. The question of condemning the carcasses of all animals affected with this disease was very important, both to the consumers of meat and to the producers of bovine animals. If there were danger of infection to man the meat should not be allowed to go upon the market, and if there were no danger, the consumer was entitled to know that the meat he was buying came from an animal affected with a serious disease. In the present condition of public opinion in the United States, the utilisation of the carcasses of animals affected even in the slightest degree would not be tolerated. Doubtless they had gone to extremes, for it was difficult to draw a definite line between cases so severe as to make condemnation necessary, and those which were so mild as not to injure the flesh in the least. So far from there being four or five per cent. of the bovine animals in the United States affected with actinomycosis, it was probable from the results of the State inspection recently established, that there were not over one or two per 1,000 among the beef cattle which arrived at the stockyards. When it was stated that from 10 to 15 affected animals were sometimes discovered in the Chicago stockyards in one day, it should be remembered that from 8,000 to 10,000 bovine animals might be received in those yards during the 24 hours.

**Mr. Thomas B. Goodall, F.R.C.V.S.** (Christchurch, Hants), spoke with great reserve as to whether such diseases were or were not contagious, as their present knowledge did not provide them with sufficient data to arrive at a conclusion. The diagnostic symptom was the yellow appearance of the nodules. It was never found without a lesion, either in the skin or mucous membrane. In two cases that he had in his museum, he found a piece of a stem of heather tightly wedged between the second and third molar teeth—in both cases the molars were "changing." It was a common thing to find lesions in the mouth at this period of life. In his cases the inflammation extended from this point into the alveolar cavities, and thence into the spongy structure of the jaw; but it might easily be imagined how such a body in such a position might scratch the tongue. Their present knowledge seemed to prove that this was a fungus which found its normal habitat in vegetable structure, and was only accidental in animals. In Dr. Fleming's work, it was stated that disease had been induced by the introduction of *Aspergille glaucus* into the system of animals. This might, therefore, be termed a contagious disease. He desired to call attention to the part played by the larvæ of dipterous insects in the great economy of life from a hygienic point of view. The mouths of these larvæ, which were reported to feed on decomposing organic matter, were so constructed that this was impossible; but, doubtless, they did feed on the lowest forms of life that induced putrefaction, and thus the larvæ of dipterous and other insects were the scavengers provided by an Omniscient Creator for keeping the number of these lowest forms of life in check. With this object in view, let them search through the life-history of the calindæ, the synphidæ, some of the musidæ, the tabanidæ, &c.

**Prof. Walley** (Edinburgh) remarked that in attending meetings of that kind, every one learned much of which he was previously ignorant. That was especially true in reference to actinomycosis, as, until to-day, he (Prof. Walley) had not been aware that internal lesions were commonly found in cattle. During the last few months the speaker

had condemned 140 carcasses, out of a total of 803 cows, slaughtered on account of pleuro-pneumonia, owing to their being affected with tuberculosis; but in not one of those cases was there the slightest evidence of the existence of external lesions, and he thought that, if any large proportion of cases of actinomycosis had existed in those cows, there would almost certainly have been some external localisation of the disease. In his view it was not a matter of much importance whether the disease was actinomycosis or tuberculosis when internal lesions existed, as under such circumstances the carcasses should unhesitatingly be condemned. Prof. Crookshank's statement showed the necessity of dealing with actinomycosis as a contagious disease, equally with tuberculosis. In reference to the contagious character of the disease, he failed to see how it could be held that the disease was not contagious, even though the contagion might only be transmitted accidentally. The previous speakers, who doubted its contagious nature, had related cases which went to show that it was transmitted by contagion. In the matter of the spread of the disease, Mr. Goodall had made a very important statement, and he (Prof. Walley) thought that much good would arise if those practitioners who frequently met with the disease would place on record the conditions under which animals in their district were kept in reference to their food, as his experience led him to the conclusion that the disease was most largely seen in poor districts, where animals were, necessarily, fed on coarse herbage. In connexion with the use of the flesh of actinomycotic animals, he (Prof. Walley) would not condemn a carcass where the lesions were localised on the tongue and skin only, as he did not think any harm would arise from the consumption of such flesh; moreover, if he or any other inspector condemned meat under such circumstances, some other professional man would be brought in by the owner of the carcass to give a contrary opinion. In this view, he thought that the sooner the disease was scheduled along with tuberculosis as a contagious disease the better.

**Mr. W. F. Barrett, M.R.C.V.S.,** (London), said that the disease was especially prevalent in Cambridgeshire and Lincolnshire, and he had found it most frequently on heavy lands. Two per cent. of the animals from Canada were said to be affected with the disease, which primarily affected the mouth and pharynx, and secondarily involved the lungs. The age of attack was usually one and a half to two years; and the animals did not appear to receive the disease from one another, but to acquire it from the same source. He asked whether the cereals grown on clay soils might not contain a larger proportion of silica, and thus injure the buccal mucous membrane, and so favour the entrance of the virus.

**Prof. McFadyean** (Edinburgh), while admitting that actinomycosis might have been confounded with other diseases, did not think that such mistakes were very frequent or important, so far as veterinary surgeons were concerned. He had been much interested in learning that actinomycosis had been mistaken for tuberculosis in Australia; for such a confusion was probably responsible for the statement that had been made regarding the prevalence of tuberculosis among cattle that had never been housed. Dr. Salmon had assured them that in the United States the sale of the flesh of animals that were affected with actinomycosis was not tolerated; but it would be interesting to learn whether the system of meat inspection in that country was adequate to insure that none of

the dead meat exported to this country was derived from animals that were the subject of actinomycosis or tuberculosis.

**Sir Henry Simpson** (Windsor), said that he lived in a district where the disease was not common. Cases which did occur appeared in animals imported into the district, but it did not die out with the disappearance of infected and imported animals; if contagious at all, it did not appear that the disease was contagious in a high degree. It was most desirable that the question of contagion and the use of the food of affected animals being consumed, should be cleared up. Whilst he was not inclined to exclude the possibility of contagion, he was quite content to recommend the consumption of the flesh of affected animals, always excluding the locally diseased parts.

**Dr. George Fleming, C.B.** (London), drew attention to the necessity of showing that there was danger to the public health or wealth before asking for legislative measures to combat the disease. There was no reliable evidence whatever of danger in consuming the flesh of animals which had suffered from actinomycosis, and the great balance of opinion was in favour of there being no risk. He deprecated raising alarm or suspicion in that direction. The malady was very prevalent in one country, extremely rare in another, and there was strong evidence that it originated in the introduction of the fungus into the body through the food. The cause was one common to both man and animals, and they had now to find out the natural history of the actinomyces outside the animal body, in order to arrest its invasion.

**Prof. Crookshank** briefly replied to the various points raised in the course of the discussion, and said he had studied the cases he had described clinically as well as microscopically. In comparing the disease with tuberculosis, it should be remembered that it was not by any means so violent as tubercle. He showed two specimens in which the naked eye resemblance to tuberculosis was very close indeed.

## Sur les Dangers que le Charbon (Anthrax) fait courir aux Ouvriers des différents Corps de Métiers.

PAR

le Profsseur M. A. Chauveau, Paris.

Dans le peu de temps attribué à l'exposition de cette grande question, on ne peut guère faire plus que de poser des principes. Ils sont simples heureusement, et laissent très facilement deviner tous les développements qu'ils comportent.

J'entre immédiatement en matière.

Un animal, atteint de charbon (*anthrax, splenic fever*) depuis son abatage ou sa mort naturelle, jusqu'au moment de la transformation ultime de ses dépouilles par l'industrie, constitue un danger pour toutes les personnes qui manipulent ces dépouilles.

Ce danger existe : 1° pour le boucher ou l'équarisseur qui opère l'abatage et le dépéçage des animaux ; 2° pour les porteurs employés

aux chargements ou aux déchargements dans les halles, marchés, ports, gares, usines ; 3° pour les tanneurs, mégissiers, pelletiers, chargés du travail de la peau elle-même ; 4° pour les ouvriers divers attachés au travail des productions épidermiques, laines, poils, crins et cornes.

Ces diverses catégories d'ouvriers sont exposés à contracter le charbon, soit externe, sous forme de pustule maligne ou d'œdème malin, soit interne, avec localisations variables suivant la porte d'entrée du germe virulent : localisations tantôt *thoraciques*, donnant lieu à ce que l'on a appelé mycose pulmonaire ou bronchique ; tantôt *abdominales*, constituant la mycose intestinale.

Le charbon *externe* est le plus connu et le plus commun.

Quant au charbon interne, ce n'est guère que depuis une vingtaine d'années que l'existence en a été mise hors de toute contestation, plus particulièrement par Münch (de Moscou) pour le charbon à localisations abdominales, et par Greenfield pour le charbon à localisations thoraciques. Ce dernier est, comme on le sait, la maladie des trieurs de laine (*Wool sorters' disease*) sur laquelle les observations du Dr. Bell d'abord, ensuite le rapport du Dr. John Spear ont appelé fortement l'attention. En raison de sa découverte relativement récente, le charbon interne paraît donc beaucoup moins répandu que le charbon externe. En France, on n'en a encore signalé que quelques cas. Mais il en est un certain nombre, à coup sûr, qui ont passé inaperçus.

Peu de contrées sont indemnes du charbon ; et là même où il est extrêmement rare sur les animaux, comme en Algérie, on a pu en constater quelques cas dans l'espèce humaine : j'ai vu moi-même à l'hôpital de Blidah, un cas de pustule maligne typique, qui a guéri, en se terminant spontanément par une escharre. Le charbon n'existerait-il pas du tout dans un pays, à l'état enzootique ou épizootique, sur les animaux domestiques de ce pays, que l'homme n'y serait pas pour cela à l'abri des atteintes de la maladie, si le commerce introduit, de contrées où règne le charbon, des matières premières nécessaires aux industries qui travaillent les peaux, les laines, les crins et les cornes.

Cette grande diffusion du charbon, sa transmissibilité à l'homme et la gravité habituelle que la maladie affecte chez lui, quand elle n'est pas reconnue et traitée à temps, rendent cette maladie très redoutable, et la designent tout spécialement aux discussions d'une assemblée internationale qui a pour mission la sauvegarde de l'hygiène publique, par conséquent la préservation de l'homme contre les atteintes des maladies infectieuses.

C'est à ce point de vue seulement que a maladie charbonneuse doit être envisagée ici, à l'exclusion de tout ce qui concerne sa pathogénie, sa symptomatologie, son traitement.

Que faut il faire pour preserver du charbon les ouvriers qui son exposés à le contracter par le fait du maniment des dépouilles des animaux morts de cette maladie ?

*Mesures propres à faire disparaître ou diminuer les cas de charbon sur les animaux.*—L'idéal serait de faire disparaître le charbon des animaux. Ce n'est pas une entreprise qui soit au dessus du pouvoir de

l'homme. On réussirait certainement à atteindre ce but, s'il était possible de faire agir tous les gouvernements civilisés, avec entente, dans l'application prolongée, tenace, persévérante de mesures de police sanitaire appropriées.

Mais si une telle entreprise se présente avec une certaine apparence un peu chimérique, il en est autrement d'une autre moins ambitieuse, celle qui a pour objectif, non de faire disparaître, mais de diminuer très sensiblement les cas de charbon qui déciment les animaux domestiques. Grâce à l'emploi raisonné des mesures de préservation ordonnées par la législation sanitaire, grâce encore à la vulgarisation de la pratique des inoculations pasteuriennes, certaines régions de la France, qui payaient jadis un lourd tribu au charbon, ont vu s'abaisser considérablement le chiffre des pertes causées par cette maladie. Or les cas de charbon sur l'homme ont diminué dans la même proportion, non seulement dans les campagnes où vivent les troupeaux, mais encore dans les usines lointaines, mégisseries et tanneries, qui s'alimentent en matières premières provenant de ces troupeaux. On peut citer une ville des environs de Paris, Saint Denis, où les seules usines dans lesquelles se déclarent des cas de charbon utilisent les peaux et les crins d'*importation étrangère*. Ces usines sont au nombre de trois. Le docteur Le Roy des Barres* y a constaté, dans une période de douze années, 49 cas de charbon, dont 10 dans un établissement où l'on ne travaille que les crins, 38 dans une mégisserie où l'on ne traite que la petite peau, et 1 seulement dans une autre mégisserie, travaillant habituellement sur des petites peaux et des peaux de mouton d'origine française. Par exception, un lot de peaux avait été exceptionnellement utilisé dans cette dernière usine, justement pendant la période où le cas de charbon s'est manifesté.

Sans vouloir donner à ces faits une importance exagérée, on peut certainement les proposer en exemple à ceux qui cherchent à préserver du charbon les ouvriers attachés au travail des peaux et des annexes de la peau. Il est certain que la pratique des vaccinations anti-charbonneuses, jointes à l'application de toutes les mesures de police sanitaire indiquées dans le cas d'apparition du charbon, rend cette maladie plus rare sur les animaux ; d'où, par une heureuse répercussion, diminution des cas qui se déclarent, sur l'espèce humaine, dans les établissements où l'on travaille les peaux, les laines, les crins, les cornes, en vue d'une transformation industrielle.

Parmi les mesures de police sanitaire auxquelles il vient d'être fait allusion, il en est une qui, à elle seule, si elle était pratiquée rigoureusement et méthodiquement, ferait disparaître, d'une manière presque complète, tous les dangers de contagion auxquels sont exposés les ouvriers divers que leur métier force à manier les dépouilles des animaux. Cette mesure, édictée dans presque toutes les législations sanitaires, c'est la destruction ou l'enfouissement des cadavres *entiers* des animaux morts

---

* Le charbon (*pustule maligne, œdème malin*) observé à Saint-Denis chez les criniers et les mégissiers. Rapport au Conseil d'hygiène publique et de salubrité du département de la Seine, 1890, et *Annales d'hygiène*, 1890, t. xxiii, p. 496.

du charbon ou sacrifiés étant en puissance de cette maladie. Si aucun animal charbonneux n'était utilisé, s'il n'en échappait la moindre parcelle à l'enfouissement, ou à la destruction, soit par le feu nu, soit par la coction, soit par les procédés chimiques, il n'y aurait plus à craindre le moindre accident charbonneux, ni sur les habitants des localités où règne le charbon, ni sur les bouchers, ni sur les équarisseurs, ni sur aucun ouvrier des industries diverses qui travaillent les peaux ou les productions épidermiques. Voilà le moyen par excellence, le seul vraiment efficace, de faire disparaître le charbon des usines exploitées par ces industries.

Malheureusement il arrive trop souvent que les cadavres sont dépouillés avant d'être enfouis, et les peaux livrées à l'industrie, en même temps que les dépouilles des sujets sains, au milieu desquelles ces peaux contaminées se trouvent confondues. Elles s'en vont ainsi porter, dans les ateliers des usines où elles sont travaillées, des germes charbonneux qui pourront infecter les ouvriers. Parmi ces derniers, combien ont été victimes des effets de cette coupable pratique des propriétaires d'animaux charbonneux ? Pour le petit gain qui en résulte, comprend-on exposé les autres, en s'exposant aussi soi-même, à être victimes d'une misérable cupidité ?

Et ce n'est pas le seul délit dont ces propriétaires se rendent coupables ! Parfois, les sujets charbonneux, reconnus malades quelques instants avant le moment où la mort naturelle surviendrait fatalement, sont saignés à la hâte et préparés comme les animaux sains. La viande en est alors livrée à la consommation, ce qui augmente considérablement le nombre des individus qui sont exposés à la contagion, par le fait de l'utilisation, défendue, condamnée par la loi, des sujets atteints de la maladie charbonneuse.

*Mesures propres à détruire les germes charbonneux contenus dans les matières premières utilisées dans l'industrie.* — Une rigoureuse sévérité dans l'application des règlements sanitaires ferait cesser ces monstrueux abus. Il faut pourtant tout prévoir : à supposer que l'enfouissement soit presque universellement mis en usage, il y aura encore à compter avec les exceptions. Là où la suppression totale des cadavres charbonneux sera decrétée et réellement effectuée, il pourra y avoir quelques erreurs commises de bonne foi ; des sujets seront abattus comme sains, au moment où ils commencent à être sous le coup d'une infection charbonneuse méconnue. De plus, certaines usines recevront peut-être encore leurs matières premières de pays où la surveillance sanitaire ne peut être sérieusement organisée. De là quelques chances de contagion charbonneuse continuant à subsister, du fait de la manipulation des peaux, des laines, des crins et autres productions épidermiques ; de là aussi la nécessité de chercher comment il est possible de faire échapper les ouvriers à ces chances de contagion.

Deux méthodes sont à examiner : 1° on peut essayer, par un traitement des dépouilles aux lieux de production, de tuer les germes charbonneux qu'elles sont exposées à contenir éventuellement ; 2° cette tentative peut être faite à l'usine où sont utilisées les matières premières que ces dépouilles fournissent à l'industrie.

*Traitement des dépouilles aux lieux de production.*—La première méthode, si elle pouvait être appliquée, donnerait des résultats plus complets que la seconde, en ce sens qu'elle préserverait non seulement les ouvriers des usines, mais encore ceux qui manipulent les dépouilles animales avant qu'elles n'arrivent à ces usines, particulièrement les portefaix qui opèrent les chargements, déchargements et transports dans les ports, gares, halles, etc.

Malheureusement on n'entrevoit pas de procédé pratique qui permette d'appliquer avec succès cette première méthode. Un tel procédé doit réaliser, en effet, les trois conditions suivantes : 1° n'entraîner qu'une dépense absolument insignifiante ; 2° n'exiger que des manipulations faciles ; 3° pouvoir être employé partout, à la campagne comme a la ville, dans les plus petites tueries particulières des moindres hameaux, comme dans les grands abattoirs publics des villes populeuses.

Mais il est au moins possible d'indiquer le principe de deux procédés, qui pourraient rendre certainement quelques services dans cette entreprise de désinfection générale des dépouilles animales livrées à l'industrie.

Le premier de ces procédés repose sur l'action microbicide qu'exerce la dessication rapide sur les bacilles charbonneux du sang frais. Exposé en couche mince à l'action d'un soleil ardent, qui le dessèche promptement, le sang perd ses propriétés virulentes, et il ne les récupère point quand on lui rend l'eau évaporée. Les bacilles asporulés du sang sont donc tués par la dessication. Et c'est bien cette dessication, et non pas l'action de la lumière solaire, qui, dans ce cas particulier, joue le principal rôle, car la destruction du bacille s'opère encore, quoique avec un peu moins de sûreté peut-être, dans les étuves sèches où règne l'obscurité.

Quant à la virulence du sang contenu dans les vaisseaux de la peau ou d'organes découpés en tranches minces, elle ne résiste pas davantage à la dessication. Il m'est arrivé souvent de faire sécher au soleil ou à l'étuve des morceaux de peau de mouton charbonneux, même des peaux entières et d'y avoir constaté ensuite l'absence de tout virulence. Je n'ai pas eu l'occasion d'experimenter sur la peau des animaux de l'espèce bovine ; mais je ne doute pas que, malgré son épaisseur, elle ne puisse être, elle aussi, assainie par la dessication. Le procédé réussit, en effet, même avec des morceaux de rate de mouton d'une épaisseur supérieure à celle de la peau du bœuf et plus difficiles à dessécher promptement.

La rapidité de l'opération importe, en effet, beaucoup à sa réussite. Si la dessication n'est pas promptement réalisée, il peut arriver que les bacilles évoluent dans les vaisseaux et forment de spores sur lesquelles la dessication, quand elle survient, n'exerce plus aucun effet. J'ai pu, une fois, constater le fait sur une rate de mouton entière, que j'ai conservée pendant plusieurs années comme une source très active de virus charbonneux.

Si donc l'industrie renonçait à l'emploi des peaux vertes, si d'autre part, dans les abattoirs publics ou privés, la peau, à peine enlevée, était de suite soumise à un traitement capable de la dessécher promptement, il y aurait les plus grandes chances pour que les dépouilles livrées au commerce ne continssent jamais le germe charbonneux.

Il semble que cette opération pourrait se faire sans embarras, puisque, à l'heure actuelle, le séchage des peaux est déjà largement pratiqué dans les annexes des abattoirs. Mais c'est un séchage lent, plutôt dangereux qu'utile, parce qu'il laisse aux spores du *bacillus anthracis* le temps de se développer. Le séchage rapide exigerait une installation spéciale et quelques frais. C'est une question à étudier par les technologistes industriels.

Le second procédé que j'ai à indiquer, pour le traitement des dépouilles dans lesquelles on voudrait être sûr que le germe charbonneux ne fut point présent, est inspiré par un principe tout autre que celui de a dessication. On ferait intervenir dans ce nouveau traitement l'action de la chaleur humide, combinée avec celle de certaines substances antiseptiques. Mais je ne le cite ici que pour mémoire. C'est dans un autre cas qu'il pourrait surtout rendre des services. La combinaison dont je vient de parler est, en effet, capable d'agir même sur les spores, d'assainir ainsi les dépouilles où des spores ont eu le temps de se développer. C'est le cas des dépouilles qui ont déjà été livrées à l'industrie. Le procédé appartient, par conséquent, davantage à la méthode de préservation qui consiste à traiter, après leur arrivée à l'usine, les matières premières exposées à recéler le germe charbonneux. L'indication de ce procédé va donc trouver sa place ci après, dans l'examem de cette deuxième méthode.

*Traitement des dépouilles à l'usine.*—Le travail des ouvriers, dans les usines, est rendu dangereux par la présence du *bacillus anthracis*, à l'état de spores résistantes, les unes cachées dans l'épaisseur de la peau, les autres adhérentes à la surface, soit du derme, soit des brins de laine, soit des crins, soit de la corne. Ces dernières sont les plus à craindre, surtout au commencement des opérations. En effet, à mesure que celles-ci s'avancent, ces spores superficielles sont entrainées ou se détruisent, ce qui explique pourquoi les premières manipulations de la fabrication sont celles qui exposent le plus les ouvriers aux accidents causés pur le germe charbonneux.

Pour mettre les ouvriers à l'abri des attaques de ce germe, les procédés indiqués par la science varient avec la nature du travail. Il faut distinguer entre les catégories d'industries. On en compte trois principales : 1° le travail des crins et des laines ; 2° le travail des cornes ; 3° le travail des peaux, surtout dans les mégisseries.

A. *Travail des crins et des laines.*—Le fait de la plus grande fréquence des cas de charbon sur les ouvriers qui se livrent aux premières manipulations de la matière première a été surtout constaté dans les usines où l'on travaille les laines et les crins. On connait la fréquence des diverses formes de charbon sur les trieurs de laine à Bradford. À Saint Denis, près de Paris, les ouvriers ou ouvrières qui, dans l'industrie des crins, sont chargés du déballage, du triage et du battage de la matière brute* fournissent également le plus grand nombre des victimes du charbon. Ce sont donc ces

* Le Roy des Barres, *loc. cit.*

premières opérations de la manipulation des laines et des crins qu'il faudrait s'appliquer à rendre inoffensives par un traitement préalable des matières. La détérioration produite par le trempage ne permet guère l'emploi des solutions désinfectantes. Mais il ne serait pas impossible d'opérer la désinfection à l'aide de la chaleur humide. L'emploi des étuves à vapeur sous pression donnerait certainement d'excellents résultats. Il a été essayé dans une usine de Saint Denis. On a dû y renoncer, parce que le procédé augmente sensiblement les frais et qu'il occasionne une légère détérioration de la marchandise. Ce n'est pas trop s'avancer que d'affirmer que ce dernier inconvenient pourrait être sûrement et facilement évité, par une bonne conduite des opérations. Quant à la dépense, il n'est guère admissible qu'elle ne puisse être rendue tolérable, dans les grandes usines où les appareils seraient surtout appelés à fonctionner, et où ces appareils pourraient être installés dans des conditions particulièrement favorables de simplicité et d'économie Il y a donc lieu de recommander de nouvelles tentatives aux usiniers intéressés.

En attendant, on devra continuer à recourir aux moyens palliatifs capables de soustraire les ouvriers chargés du triage et du battage à l'action des poussières qui se dégagent dans ces deux opérations. Pour cela, on s'est bien trouvé partout de l'installation d'aspirateurs qui emportent les poussières à mesure qu'elles se dégagent pendant les manipulations exécutées à proximité des bouches de ces aspirateurs, soit audessus, soit audessous. Les patrons soucieux de leurs devoirs envers leurs ouvriers ont spontanément installé ces aspirateurs dans leurs usines : il serait bon que tous, sans exception, fussent obligés d'y avoir recours.

B. *Travail des cornes.*—Les usines où l'on travaille les cornes ne nous retiendront guère. Ici tout est facile, si on le veut, car la matière première se prête à toutes les opérations de la désinfection. Traitement par l'eau surchauffée, par la vapeur sous pression, par les procédés chimiques, etc. : tout convient, tout peut être essayé.

C. *Travail des peaux.* — C'est dans les usines où se fait le travail de la peau elle-même qu'il est le plus difficile de traiter les matières premières pour les rendre inoffensives, car elles sont d'une part très altérables, d'autre part extrêmement encombrantes. On ne peut plus penser ici à l'emploi de la vapeur sous pression. De plus, les manipulations qu'exigerait un traitement chimique prélable augmenterait considérablement les frais de fabrication. Aussi, pour éviter ces frais d'une opération spéciale, certains mégissiers ont-il songé à profiter d'une des opérations nécessaires de la fabrication elle-même, le *trempage.* Au lieu de faire cette opération dans l'eau ordinaire, on peut ajouter à l'eau une substance microbicide, capable de détruire la végétabilité des spores du *bacillus anthracis.* M. Pasteur a conseillé l'essence de térébenthine. Malheureusement à la dose que l'eau en peut dissoudre, cette substance est peu active ; de plus, elle nuit à la qualité des produits ; aussi son emploi n'a-t-il pas été continué.*

---

* Le Roy des Barres, *loc. cit.*

Mais il est certainement possible de réussir en utilisant d'une autre manière l'opération du *trempage*. Je rappelerai ici un principe établi d'après les résultats de plusieurs séries d'expérience que, M. Arloing et moi, nous avons faites à Lyon. Ces expériences ont démontré qu'en combinant l'action de substances antiseptiques, impuissantes à modifier l'activité de certains agents pathogènes, avec l'effet d'un chauffage également impuissant à atteindre ces agents pathogènes dans leurs propriétés essentielles, on réussit pourtant à en détruire la virulence ; et cela arrive même avec les agents qui comptent au nombre des plus résistants que l'on connaisse, comme le vibrion septique sporulé. Or cet agent ne résiste pas à la température de + 40° au contact un peu prolongé de l'acide phénique employé aux titres usuels de l'usage chirurgical. Naturellement cette influence destructive croit avec l'élévation de la température du chauffage. Dans le cas particulier qui est considéré ici, la température pourrait certainement être porté sans inconvenient pour la matière première jusqu'aux environs de + 58° à + 60°.

Donc l'action combinée d'une chaleur modérée avec celle d'une substance microbicide ajoutée à l'eau où doit s'opérer le *trempage* des peaux, dans les mégisseries, doit constituer un procédé de désinfection très efficace, qui mérite d'être étudié pas les chefs d'industrie.

Tels sont les procédés palliatifs auxquels il conviendrait d'avoir recours pour dinimuer, sinon pour faire disparaître, les dangers de contagion auxquels sont exposés les ouvriers employés dans les industries qui exploitent les matières premières provenant des dépouilles des animaux domestiques.

*Addendum. — Alimentation avec des viandes charbonneuses.* — Dans toute cette etude, j'ai laissé de côté les dangers qui résultent de l'usage alimentaire de la viande provenant d'animaux charbonneux. Ce point est ici hors de cause. Pourtant il se rattache étroitement au sujet qui vient d'être traité. En effet, comme je l'ai déjà dit, ce n'est pas seulement pour l'utilisation des dépouilles des animaux charbonneux que ces animaux sont abattus par effusion de sang, lorsqu'on les voit sur le point de mourir ; la viande elle-même est parfois utilisée ; on cherche à la faire consommer et, de cet acte vraiment criminel, il est résulté des accidents graves d'infection charbonneuse : pustules malignes, provenant de l'inoculation externe pendant la manipulation de la viande fraiche ; charbon interne déterminé par l'ingestion des germes virulents. Quoique ceux-ci n'existent dans la viande qu'à l'état de baccilles facilement détruits par la cuisson et par les sucs digestifs, il arrive que quelques-uns échappent à cette double action destructive et infectent les sujets. Des exemples absolument indéniables de ce mode d'infection ont été constatés. Raison de plus pour se montrer sévère dans l'édiction des mesures qui ont pour but de mettre l'espèce humaine à l'abri du charbon.

*Conclusion.*—Ces mesures, en résumé, doivent tendre à faire disparaître des diverses contrées le charbon des espèces animales. S'il n'y avait plus d'animaux charbonneux, l'homme ne serait plus exposé à contracter le charbon, et il n'y aurait besoin de prendre aucune précaution pour l'en préserver.

Il se trouve justement que la principale de ces mesures, la plus efficace, est aussi celle qui, dans le présent, contribuera le plus à soustraire l'espèce humaine aux dangers d'infection charbonneuse. Cette mesure fondamentale, c'est la destruction, ou, à défaut de la destruction, l'enfouissement méthodique des cadavres *entiers* des animaux charbonneux, avec destruction ou désinfection de tous les objets que leurs déjections auront pu souiller. Il est bien évident que, si ces animaux charbonneux étaient immédiatement détruits après leur mort, ils ne feraient plus courir aucun danger à l'homme. C'est, sous une autre forme, le même truisme exprimé tout à l'heure : il n'est pas inutile de le répéter, pour appeler l'attention d'une manière tout particulière sur cette mesure de la destruction ou de l'enfouissement, c'est à dire sur le moyen héroïque qui est seul capable de mettre à l'abri du charbon les ouvriers des industries du crin, de la laine, des cornes, des peaux.

Prescrite dans tous les pays civilisés, cette mesure n'est peut-être nulle part convenablement appliquée. Il importe de travailler à sa propagation, par des instructions populaires, largement répandues, dans lesquelles on démontrerait aux cultivateurs que la destruction ou l'enfouissement des animaux charbonneux sauvegarde leurs propres intérêts. L'effet de cette propagande devrait être renforcé par une bonne organisation des services de surveillance sanitaire et par une application sévère des pénalités contre les délinquants.

Quand on met la simplicité et l'efficacité de cette mesure en présence de la complexité, des difficultés d'application et de l'insuffisance des moyens pallatifs qui pourraient être employés dans les abattoirs ou les usines, on voit de suite où se trouve la supériorité. D'un côté, manipulations préalables s'appliquant à des masses énormes de matières premières saines, pour atteindre quelques parties contaminées, manipulations coûteuses et d'application encore incertaine ; de l'autre côté, mesure simple, quasi gratuite, absolument sûre dans ses résultats, s'appliquant, aux lieux de production du bétail seulement, à un nombre excessivement restreint de sujets, profitant au producteur lui-même, présentant enfin cet autre avantage moral d'incomber à celui chez qui et souvent par qui le mal a été créé : comment pourrait-on hésiter sur le parti à prendre ?

Demandons donc l'extension et l'application rigoureuse de la destruction ou de l'enfouissement des cadavres d'animaux charbonneux : c'est le seul moyen vraiment pratique de protéger les ouvriers de nos usines contre les atteints du charbon.

## Anthrax and its relations to Workers in various Trades.

BY

W. Duguid, F.R.C.V.S., Assistant Inspector of the Veterinary Department of the Board of Agriculture.

The disease known as anthrax, splenic apoplexy, charbon, miltzbrand, etc. in animals, and as malignant pastule, contagious carbuncle, and wool

sorter's disease in man, is not only one of great importance on account of its transmissibility from animals to man, but it is otherwise of considerable interest from a scientific as well as a practical and financial point of view.

Scientifically it is most interesting on account of its being the first affection of man or animals in which a micro-organism was proved to be the cause. The organism on which anthrax depends was the first pathogenic microbe whose life-history was studied in the laboratory by means of the microscope and the methods of cultivation on nutritive media now in use. The study of anthrax may be looked upon as the starting-point from which the germ theory of transmissible diseases was evolved and the application of bacteriology to pathological research begun.

With this reference to the connexion between anthrax and bacteriology, we will now take up the importance of the subject from a practical and financial point of view. Anthrax is of much practical interest to the medical man, who may be called upon to diagnose and treat cases of the disease when it has been transmitted from animals or animal products to man. To the veterinary surgeon, who may be called upon to decide whether an animal is affected with or has died from anthrax, and to advise as to what means should be adopted to prevent the contagion spreading, a practical knowledge of the disease is absolutely necessary. To the agriculturist and stock-owner, whose animals are attacked and often die in considerable numbers, I need scarcely say that anthrax becomes often a subject of very considerable financial importance.

Before dealing with the question of prevention, let us consider the geographical distribution, history, animals most susceptible, and means of transmission of this disease; and we shall then be better prepared to discuss the more practicable and reliable means of prevention.

*Geographical distribution.*—Anthrax is one of the most widely-distributed diseases of animals, being known over the entire surface of the globe; attacking not only animals in a state of domestication, but often destroying large numbers living in their natural or wild condition. It has been described by travellers in all parts of the world, and is known in Lapland and Siberia as well as in the tropics and in more temperate regions. No country or climate is known to be free from it, although it is more prevalent and destructive in some countries than others.

*History.*—Anthrax has no doubt been known from the earliest times, and according to several acknowledged authorities it constituted one of the plagues of Egypt as described by Moses, when there was "a breaking "forth with blains upon man and upon beast throughout all the land of "Egypt," . . . "upon the horses, upon the asses, upon the camels, upon "the oxen, and upon the sheep." The fact that the animals mentioned are most susceptible to anthrax seems to strengthen the view that the destructive scourge thus described was a serious and extensive outbreak of anthrax. In the early and middle ages, writers mentioned a devastating plague among animals spreading from them to man. This was probably anthrax in some instances, though possibly not in all.

There seems good reason to believe that, until the end of last century, the different forms of anthrax were often described as distinct diseases, while other maladies of a less virulent nature were often accepted as anthrax.

It was not, however, until the discovery of the bacillus anthracis was made that the true nature and cause of anthrax became known.

*Discovery of the bacillus anthracis.*—In 1855, Pollender made known his discovery that in the blood of animals recently dead from anthrax, large numbers of fine, rod-shaped bodies existed, which in their micro-chemical behaviour were apparently of a vegetable nature. Independently of Pollender, Branell, in 1857, found the same organism in the blood of men and animals which had died from anthrax, and from that date a new era began, not only in the study of anthrax but of other contagious or transmissible diseases.

Prior to this discovery anthrax had been generally looked upon as more or less of a malarial affection, and its appearance was generally considered to be due either to some condition of the soil, or of the animals affected, brought about by climatic or other influences. Even after the bacillus was discovered many doubted whether this organism was the cause or the result of the disease, but experimental inoculation of susceptible animals with blood containing the bacilli satisfactorily proved the contagious nature of the disease.

While the investigations into the nature of anthrax were being conducted on the Continent of Europe by Davaine, Delafond, Gerlach, and others, both prior to and for some time after the discovery of the bacillus, no mention is made in our veterinary literature of the existence of the disease in England; and if it did exist it must have either escaped the notice of the veterinary profession or have been described under other names. The first time I believe splenic apoplexy, as anthrax was at first named in this country, was recognised, was in the winter of 1858–59 on a farm in the north of England, where some animals had first died from it in 1857. Much about the same time it was recognised in the Valley of the Yeo, near Ilchester in Somerset, and from inquiries then conducted no doubt can be entertained that it had existed there in 1855, if not earlier. It has now been recognised throughout the whole of England and other parts of the United Kingdom: nay more, many districts must now be considered as permanently infected.

*Varying susceptibility of different kinds of animals to anthrax.*—As the main object of the present discussion on this disease is the protection of the workers in certain trades and manufactures, it is evident that the greater or less susceptibility of different animals to the infection of anthrax may have a material bearing on the protective measures to be adopted, according to the frequency with which the flesh and other products of the more susceptible are utilized for food and other purposes.

Anthrax is primarily a disease of animals, and more particularly of ruminants. They are most susceptible to its infection, and in them it proves most fatal. Few, when once attacked, recover. Other herbivorous animals, such as the horse, rarely suffer, but when attacked usually

die. It was for a long time believed that omnivorous animals (pigs were not susceptible, but Dr. Crookshank proved experimentally that they took the disease readily enough, when either inoculated with the blood or fed on the flesh of animals which had died from the disease. But although susceptible to infection, pigs more frequently recover from anthrax than either cattle or sheep. Out of 210 pigs affected in Great Britain last year, 117 or over 55 per cent. recovered, whereas there were only 27 or about 10 per cent. recoveries reported out of 253 cattle attacked, and the whole of the 72 sheep affected died.

The carnivora enjoy a greater immunity from anthrax than the other mammalia. It is seldom they become affected, even when inoculated or fed experimentally on anthrax flesh. The susceptibility of different kinds of animals to anthrax here given is based upon the statistics collected in Great Britain during the past four years. Out of 1,913 animals attacked, over 70 per cent. were ruminants, 28 per cent. were pigs, and less than 2 per cent. were horses, while in only a few returns were dogs or cats mentioned. From the above, it is evident that anthrax most frequently affects the animals whose flesh is used for food, and the other parts of whose carcasses are invariably used for trade purposes.

*Transmission of the disease.*—Anthrax differs from other contagious disease in this respect, that it is seldom, if ever, transmitted from the living diseased animal, but from its carcass after death. There are three modes by which the bacillus anthracis or its spores may gain an entrance into the body:

1. Direct inoculation through a wound or abraded surface.
2. Ingestion with some article of food.
3. Inhalation into the lungs of the spores in the dust which often exists in dried animal products.

In the case of animals, the second mode of transmission seems most common, for the post-mortem lesions are most frequently met with in the digestive organs, but it is seldom that the article of food which has been the infecting medium is satisfactorily made out; and I may here mention that although we know the life history of the bacillus, and the manner in which the spores are developed in laboratory cultivations, we do not know all the conditions under which these spores retain their vitality apart from animal bodies and nutritive-cultivating media.

The frequency with which anthrax recurs in the same fields and pastures, and after long intervals, points clearly to a prolonged vitality under circumstances and conditions but little understood. It is true Pasteur has pointed out that the spores may be brought to the surface of the soil from buried carcasses by earth-worms,* but there appears need for further experimental research in this direction, to explain why the disease appears several years in succession in the same fields and then disappears for a season or two, only to recur again in a form as virulent as ever.

---

* This has been denied by Klein and experimentally disproved by Koch, *Mittheil. a. d. k. Gesundheitsamte,* 1881. [Ed.]

In man the first and last modes of infection are most common. Men handling the fresh carcasses or remains of animals recently dead from anthrax often get inoculated through wounds or abrasions on their hands or arms, and they may also get inoculated by the sting of flies, or other insects, which have been in contact with the viscera, &c., of anthrax carcasses.

*Wool-sorter's disease.*—With regard to the infection of human beings by the inhalation of anthrax spores, this occurs almost invariably in those connected with wool-sorting. The form of disease known as "wool-sorter's disease," appears to have been first observed and described soon after foreign wool and mohair became regular articles of trade in this country. For long its true nature was not known, and it is only about 10 or 12 years ago that Spear first demonstrated that wool-sorter's disease was really anthrax, communicated through the spore-containing dust inhaled into the air passages and lungs.

*Different forms of anthrax.*—As might be expected this disease presents certain modifications or peculiarities in its symptoms according to the mode of infection. Thus we find when direct inoculation takes place, it assumes the form of malignant pustule in man and in animals. We also sometimes meet with cases of anthrax with external manifestations.

When infection takes place by ingestion, the disease runs its course very rapidly, and the post-mortem lesions are found in the intestines, spleen, and other abdominal organs. When inhalation has been the means of infection, we find the lungs and air passages affected, as already mentioned in woolsorter's disease.

*Occupations in which infection with anthrax most frequently occurs.*—In referring to the means by which anthrax is transmitted to man, I have already indicated to some extent the people most likely to contract the disease.

1. Veterinary surgeons, butchers, or knackers' men engaged in making post-mortem examinations, dressing or skinning the carcasses of animals which have died from anthrax.
2. Men in tan-yards and others, handling the skins or other parts of the carcasses in the course of their ordinary work.
3. Wool-sorters.

*Prevention.*—As anthrax is always communicated to man from animals or animal products, the first step towards its prevention in man would be, if possible, to get rid of the disease in animals.

Legislation naturally occurs to every one as a means of checking the spread of contagious animal diseases by preventing the movement of those affected and their association with healthy animals; but, as already stated, anthrax is not communicated by the living animal, and therefore legislation for this disease must take a different form to that suited for other diseases.

By an Order of Council dated September 1886, anthrax was first legislated for in this country, and this order is mostly permissive in character. To give notice of the existence of the disease is optional on

the part of the owner of an animal suffering from, or suspected of, anthrax. When notice is given to the local authority of the existence of anthrax in any place, they are empowered by this Order to make regulations as to the movement of the affected or suspected animal, or of those in contact with it, or of the fodder, litter, or other things with which it may have been in contact. The disposal of the carcasses of animals which have died of anthrax by burying them deeply and covering them with quicklime or destroying them by heat or chemical agents is, however, compulsory on the part of the local authorities, as well as the cleansing and disinfection of the premises where the diseased animal was kept or died.

After four years or more of this form of legislation, we find that very little progress has been made in reducing the amount of anthrax among farm stock in this country. In 1887 there were 649 animals affected in Great Britain, in 1890 there were 543, or 106 less; but so far as legislation is concerned, we have no guarantee that there may not be a material increase at any time. The question of what legislation should be adopted in connexion with anthrax in animals is open for discussion.

*Inoculation.*—Protective inoculation, by means of an attenuated or modified form of virus, has been adopted in France with marked success. Pasteur has successfully carried out this attenuation of the anthrax virus in the laboratory, and produced a protective inoculating material which gives immunity from natural infection if not for the life of the animal, at least for a considerable time.

A general adoption of any system of compulsory inoculation in this country could not be justified, considering that last year out of 6,508,632 cattle only 253 were attacked, and out of 27,272,459 sheep only 72 were affected.

In addition to this, the results of the few experiments on protective inoculation which have been conducted in this country with this attenuated virus have not been of a very satisfactory or encouraging nature; and before it could be adopted on farms where the disease frequently occurs, some modification is required in the process of preparing the protective material. The ordinary veterinary surgeon has neither the time nor the appliances at hand, had he even the manipulative skill, to carry out the work. What is wanted is some ready mode of attenuating the virus which the veterinary surgeon could adopt without delay when an outbreak occurs in which inoculation is considered expedient or advisable.

The result of some experiments conducted at the Brown Institution by Dr. Burdon Sanderson, in the summer and autumn of 1878, pointed to a possibility of modifying the anthrax virus so as to obtain a protective material by passing it through the bodies of small susceptible rodents. It was noted in one series of experiments on anthrax, in which guinea-pigs were used, that after some two or three removes from the cattle the inoculated guinea-pigs lived some hours longer than those inoculated direct from the cattle. This suggested some modification or attenuation of the virus, and it was resolved to inoculate back into a bovine animal

and watch the result. This was done, and a six-months-old calf was inoculated subcutaneously from one of these guinea-pigs. At the end of 24 hours the calf showed signs of illness with rise of temperature, by the end of 36 hours the temperature had risen to 106°F., the animal could scarcely walk without staggering and seemed dying, but by the end of 48 hours after the inoculation signs of recovery appeared and the temperature had fallen somewhat; the recovery was complete after the lapse of another two or three days. At the end of a week the same animal was again inoculated, but the second inoculation produced no decided result beyond a slight rise of temperature for one day. Similar inoculations were made in another animal with a like result.

Dr. Greenfield continued this inquiry further, and afterwards this mode of obtaining a protective inoculating material was adopted by Professor Roy and put to a thoroughly practical test, when he succeeded in arresting a most extensive and serious outbreak of anthrax among cattle in South America.

The recent experiments carried out by Hankin, in which he found that the serum of rats' blood and also a proteid he prepared from rats' spleen counteracted the effect of anthrax inoculation in mice and prevented death, seems also to be a step in the right direction, and one to be still further pursued.

Before leaving this subject of preventive inoculation for anthrax I would remind you that there is a great probability of its value being over-estimated. Anthrax very often, even in serious outbreaks, ceases suddenly without inoculation being adopted. I have been consulted about outbreaks which ended abruptly, and if I had inoculated in these cases I should no doubt have given the credit to the inoculation, when really the cessation of disease was due to other causes.

*Disposal of carcasses.*—No attempt should ever be made to utilise any part of the carcass of an animal which has died of anthrax. In trying to obtain salvage for such carcasses, great risk is run of the disease being communicated not only to man, but also to other animals. On two occasions I have seen the most serious consequences from this practice, which, I believe, is only too common in this country. In one case where an animal died, and in the attempt to dress the carcass for human food blood and offal got spread about, no less than 46 cattle died in less than three days. In the other case, from a similar cause, 39 cattle died in four days, and—what was more serious—three men got infected and nearly lost their lives by direct inoculation.

The destruction of such carcasses by burning, if practicable, would be the best means of disposing of them, but such a course cannot often be carried out. Deep burial of the carcass, whole, in some secluded spot which other animals do not frequent, and in some retentive subsoil, at a distance from any brook or source of water supply, is the most practical way of disposing of them, especially if covered with a quantity of quicklime or other disinfectant capable of destroying the infective power of the organism.

In towns where neither the burning nor burial of the carcasses can be carried out, we are obliged to dispose of them by sending them to the

knackers' yards. This is always attended with a certain amount of risk, because in the ordinary course the skins are utilised, and other parts of the carcass, after boiling, are often used as food for other animals, including pigs.

*Prevention of anthrax in man.*—Legislation in this country has not been attempted with this object in view, and if it were, in order to render it of practical value, it would require to be of so severe and restrictive a character as would be impracticable to carry out. Nothing short of the absolute prohibition of the use for trade purposes of the remains of animals (hides, hair, wool, &c.) which had died from anthrax either at home or abroad, or their disinfection by some process which would render them harmless, would be effective.

To carry out the first would be impossible unless the importation of such animal products was prohibited from every country in the world, as none of these are free from anthrax. I need scarcely say that such a measure could not be carried into effect. With regard to products from home-bred animals, such legislation could hardly be carried out.

We must therefore fall back on the alternative of disinfection, and here we are met with two difficulties:—

(1.) What modes of disinfection can be applied to these animal products which will not injure them for trade purposes? and (2.) To be absolutely safe, not only imported animal products, but also those of home production must be submitted to the methods of disinfection selected.

With regard to the best modes of destroying the vitality of the spores of the anthrax bacillus, so as not to injure animal products for trade purposes, more than one must be selected. The high temperature which might be permissible in the case of wool would completely destroy hides, while chemical disinfectants which would not injure the hides might seriously injure or destroy wool for manufacturing purposes. A series of experiments on the best means of destroying the vitality of the anthrax bacillus might prove of considerable value in this direction.

Any compulsory disinfection of this kind would of necessity, to be of value, have to be conducted under supervision of the Government, and would prove a serious undertaking.

## DISCUSSION.

**Dr. Samuel Lodge, Junr.** (Bradford), observed, in reference to the history of woolsorters' disease, that his father, when asked by the Woolsorters' Society to make an investigation into the cause of death of a sorter, saw the anthrax bacilli in the blood, but not knowing them as bacilli, he described them as "very fine fragmentary hairs," being, of course, entirely ignorant of the existence of such a thing as a bacillus. Sorters knew that the fallen fleeces were chiefly dangerous, because the animals had died of splenic fever. The present regulations, although they had been the means of almost stamping out the disease, were not efficient. Experiments had been made by Dr. Wurtz of Paris and himself as to the best means of disinfecting materials infected with anthrax. Steam

at high pressure and temperature of at least 120° C. should be used for a period of time to be determined experimentally. They thought that the same regulations should be applied to rag warehouses, since they had had a fatal case in a rag-picker. The bacilli were found in tissues of the woman after death, and it was subsequently shown by experiments that the spores of the anthrax bacilli were contained in the dust of the room.

**Dr. George Fleming, C.B.** (London), referred to the great antiquity of anthrax. The wool, hair, and skins were the chief sources of infection of workpeople. This danger could be avoided by freeing these products from the germs, by processes which were now well known. Animals which died from anthrax should be burned whenever possible, not buried.

**Dr. T. W. Hime** (Bradford) said the difficulties of preventing anthrax would be understood better if the conditions surrounding the utilization of various animal products were borne in mind. In Bradford the disease was really not common, so far as reported cases went, but he thought there was reason to believe that there were many more than were recognised. The appearance of large numbers of deaths from pulmonary disease among men in the prime of life terminating in a very few days, suggested something not like ordinary pulmonary disease. It sometimes even happened that the wives and children of persons manipulating infected wools contracted the disease, no doubt through contact with a person coming from the infected workshop. Attention first began to be drawn to the existence of some serious and unfamiliar source of disease among woolsorters after the introduction of alpaca by the well-known firm of Sir Titus Salt. It was observed that deaths occurred under peculiar circumstances, but the cause was mysterious. It had been mainly owing to the exertions of Dr. J. H. Bell, of Bradford, that the disease had been shown to occur among sorters of various kinds of wool, and to be identical with anthrax or charbon. It was almost limited to wools which came from warm climates, *e.g.*, alpaca, mohair, camel, &c. The wools were of a dry character, containing but little "suint" or natural grease, and arrived in Bradford in a very dirty state, except when accidentally wetted en route. It was common to find the "bales" (large packets weighing some cwts.) containing dirt of every kind, dung, gravel, even bits of skin torn off with the wool, and small clots of blood. Even in bales of the finest material "bumps" of such dirty wool were frequently found, and were recognised as "fallen," or wool which had been torn from dead animals, and not shorn from the living. When bales of these dry wools were opened, thick clouds of dust rose, enveloping the opener and filling the room. As a rule, there was nothing which could be called a stench, even from bales proved to contain anthrax virus. These wools were of great value, and a bale weighing 7 cwt. would be worth a large sum of money in the rough. This high value made the question of disinfecting them, with a view to destroying any virus, really or possibly present, a serious one from a commercial point of view; while the enormous quantities imported made the practical problem a very difficult one. At present very little was known as to the effects of any method of disinfection of such materials. In Bradford a code of regulations had been adopted with a view to diminishing the risks from such materials. They were originally drafted by him (Dr. Hime), but were subsequently modified by independent committees representing the sanitary authority, the

employer, and the employed, and later by a conjoint meeting of these committees. Finally, a coroner's jury, in their verdict as to the death of a woolsorter from anthrax, expressed its opinion that the regulations ought to be carried out and be kept posted up in every sorting room. This recognition of the value of the regulations (imperfect though they might be) was a matter of vast importance. It involved an authoritative announcement that certain materials were dangerous to those manipulating them, and threw on masters and men the serious responsibility to carry out the regulations which competent persons had declared to be necessary, and which were within the knowledge of both parties. Should a death be caused in open violation of these regulations, it would be for the jury to say whether a charge of manslaughter did not lie against the offending party. The more important details in the regulations were :—

1. The definition of certain kinds of wool as "noxious."

2. The particularising of the danger from dust arising from certain wools (mohair, camel, Persian, Cashmere, and alpaca), and the necessity of measures being adopted to minimise their danger by the employment of suitable means, especially "fans" so constructed as to draw the dust downwards from the person of the workman.

3. The necessity of dealing specially with noxious wools before they were given to the sorter to be manipulated, in such a way as to protect the latter.

4. The prohibition of taking meals in the workroom.

5. The necessity of keeping the sorting-room clean, well ventilated, and disinfected, and free from stored goods.

He thought that efforts should be made to make it known at the points of exportation that responsibility of a serious kind was attached to the forwarding of infected material, and that whenever material was delivered to a workman to manipulate, which there was reason to believe was probably dangerous, the workman should be made aware of it. He felt satisfied that the regulations of 1884 had led to a great general improvement in the conditions under which woolsorters worked. Now that the regulations had been in force so long, and all parties had proved that good had been derived from them, and that none of the gloomy forebodings as to their interference with trade, &c. had been realised, they might with great advantage be revised, and some of his original proposals, which were excised, should be re-inserted, more particularly those regarding the danger of men wearing, out of the workroom, the overalls they wore while engaged in sorting the disposal of refuse from the sorting-rooms, etc.

**Professor Brown, C.B.** (London), said that one of the objections urged lately against protective inoculation seemed to deserve a little consideration. It had been suggested by practical men that the inoculation of some food animals, many of them a short time before they were slaughtered, was by no means free from danger. They had to consider the delicate nature of the virus, which was not sent out by Monsieur Pasteur excepting upon a distinct understanding that it should be used within a certain number of hours. There was a great risk of their arriving at entirely wrong conclusions as to the effects of its operation in this country. It was not uncommon for a single animal in a herd o be attacked, and for there not to be another case

on that farm for years. The fact that the disease often ceased after a few attacks was certainly opposed to the idea of introducing to the remaining animal systems a virus of somewhat doubtful character at the time at which it was used. The question of the destruction of diseased carcasses by burning had not escaped the notice of the Board of Agriculture, nor, previously, of the Privy Council. The objections against burial were and had been recognised; but the objections against burning were ten times greater. In the first instance, not one place in a thousand would establish the machinery necessary for burning carcasses whole, and it would be necessary to adopt the most objectionable practice of cutting up the carcass before burning, whereas in burying, the carcass might be buried entire. Dr. Klein had shown by experiment that the carcasses of smaller animals, when buried without their tissues being exposed to the air, lost all trace of bacilli in a few days. One would not be disposed to recommend the system of burning, unless there were means of putting the carcasses into the destructor uncut. There were very few of such machines capable of taking uncut carcasses—certainly not of those of the horse or the ox. He did not believe there were ten in the country. With reference to the question of woolsorters' disease, he quite agreed with Dr. Hime, that certain precautions might diminish the infection. It would be interesting to the Congress to know that some time ago ingenious suggestions were made to the Privy Council on this subject, and especially one which insisted that inspection of wool should take place at the point of landing, so that the woolsorters might no longer incur the risk of fatally inoculating themselves during the process of sorting. As to this, by the way, he understood that their delicacy of touch was so great that they could distinguish between wool clipped from the living animal and that taken from the carcass. Supposing the suggestion had been carried out, it would simply have had the effect of transferring the risks of the woolsorter to the officers employed by the Customs. In fact, by the adoption of that scheme, they might find outbreaks occurring round every port in the kingdom.

## Veterinary Hygiene.

BY

FRED SMITH, M.R.C.V.S., F.I.C., Professor in the Army Veterinary School, Aldershot.

In discussing all questions at a Congress of this kind, we have to show, firstly, how much we know of the subject, and secondly, how much has yet to be learned. In no section could any question of Veterinary Hygiene be better taken up than in this one; it is proved beyond doubt that there are diseases transmissible from animals to man, and certain preventive measures naturally suggest themselves. Now, these preventive measures, so far as animals are concerned, fall entirely within the province of the veterinary surgeon, and it will be my duty in this

paper to show how far we, as a profession, are prepared for the responsibility thus thrust upon us, and what measures we adopt to ensure that in the matter of State medicine we are endeavouring to keep pace with the progress of science.

The conditions under which animals live in this country cannot be considered satisfactory; it is true that in many respects we are ahead of other wealthy nations, but, taken all round, it is difficult to make the horse and stock owner place a real value on those laws which govern the health of the animal community, or to estimate, when he is not immediately the sufferer, the cost of disease. Parkes' axiom that nothing is so costly as disease, and nothing so remunerative as the outlay which augments health, applies with as much force to the lower animals as to mankind.

To establish our position it is necessary that I should briefly review the aims and scope of Veterinary Hygiene, and endeavour to show in what a variety of spheres its usefulness may be demonstrated.

Taking firstly, then, the most natural sequence of events, viz., breeding, we find that though in some cases considerable care is taken in the selection of animals for this purpose, yet a great deal in this direction still remains to be done. It appears very difficult for the ordinary lay mind to grasp the fact that "like breeds like," the consequence being that in one class of animal, namely horses, the amount of hereditary unsoundness is simply appalling. Veterinary surgeons of great experience in the examination of horses for soundness will tell us that a really sound animal is the exception, and that the unsound one is so common that we have been compelled to adopt a certain paradoxical formula in describing an unsound horse which is fit for work, viz., as practically sound. The term is a loose one scientifically, and logically incorrect, but as a saving clause it is essential so long as want of care and more thorough supervision in the breeding of horses is withheld.

The whole country should be indebted to those various societies, prominent amongst which is the Royal Agricultural, which have endeavoured to improve the soundness of our equine produce; their work in this direction is a grand one, though the results are of necessity slow in developing themselves. We must reach not only the big breeder but the small one also; we must teach the small farmer and horse owner that when a mare is no longer fit for work, through incurable lameness or other cause, that she is a most unsuitable subject to breed from; and yet this exactly represents our case at the present day. No longer fit to work through disease, the mare is used to breed from, and the results are, as may be imagined, sapping our vitality as a horse-breeding nation, and throwing on the market worthless and unsound animals.

Our position in the matter is very clear. It would be too much to expect legislative interference in the matter of either horse or mare, the remedy lies with our horse societies, and in instructing the masses; the former are keenly alive to their responsibilities in this matter, the latter we can only hope to deal with through the civilizing process of education.

The position of Veterinary Hygiene in respect to the breeding of animals is this,—we can impress the necessity of using sound adult stock; we can teach the conformation best suited for different kinds of work, what to accept and what to reject; we can urge the necessity of breeding from mature animals, of feeding the pregnant animal liberally on that class of food best suited to the requirements of mother and fœtus, we can show the necessity for protecting the larger animals which are in a pregnant state from wind and weather, and for placing them under hygienic conditions; we can point out how neglect in this direction is a common cause of abortion, how the latter may also be produced by certain kinds of food, emanations from drains, &c. We can carefully direct the feeding of the young stock according to their requirements, impress the urgent necessity for liberal attention in this matter, and the disastrous consequences of systematic starvation in the ultimate stock; in fact, the advice which we can render to the breeder of all classes of animal is almost endless, and in course of time it must make itself felt. At present our horses are consumed by unsoundness of limbs and wind, our cattle by tuberculosis. The application of the laws of hygiene would in ten years revolutionise the existing state of affairs.

During the growth of young animals, we can advise about their feeding, the nature, quality, and quantity of food required, and in the case of cattle, as to their fattening and general care. The training and management of the young horse is a point on which we are especially capable of affording useful information, as there can be no doubt that much harm is done to young horses at this time from being worked beyond their strength.

In this way we can follow the animals until they arrive at adult life; we now deal especially with their food and feeding, water and watering, ventilation and drainage of their stable or buildings, the regulation of their work, the fitting of their saddle and harness so as to prevent injury and enable them to perform their work with the least expenditure of force; the care of the feet and prevention of disease by attention to shoeing. Advice respecting the clipping of their coats in winter, their grooming and general cleanliness, the prevention of stable accidents by the adoption of due precautions suggested by experience. In this way our unremitting care and attention will ward off disease and accident, and extend their useful lives.

But our advice does not end even here; during epizootics we can prescribe the best methods of avoiding infection and reducing loss amongst those attacked; in the case of infectious diseases we can stay their spread by the adoption of methods of isolation, destruction, and disinfection, and we can advise on the best methods of disposing of the bodies of diseased animals so as to prevent further spread of infection.

Apart from the above, there are special branches of Veterinary Hygiene (one of which it will be the object of this paper to expound) which are of public interest and utility. I have alluded to the stamping out and suppression of infectious diseases, which legitimately falls under this section. Veterinary State Medicine received its impetus in this

country from the fearful visitation of the cattle plague. Our insular position affords us exceptional facilities for keeping away from these shores diseases which on the continent of Europe are enzootic; on the other hand, the enormous supplies required by this country, necessitating the importation of cattle from both Europe and America, expose us daily to infection. Our port inspection and quarantine methods are entirely to be thanked for the exclusion of such diseases as cattle plague, sheep-pox, and lung plague. Owing to the difficulty of diagnosis, cases of the latter no doubt escape detection, but on the whole our port inspection is excellently performed, and the public has but a faint notion of the value of the work carried out and the jealous watch unremittingly kept over the agricultural and public welfare.

Meat and milk inspection is a special branch of Veterinary Hygiene; we do not for one moment pretend to say what food is suitable for human consumption, this obviously falls to the share of medical officers of health, but we can speak with certainty as to the condition of the animal furnishing the meat, and the state of health of the cow. It is clear that here Human and Veterinary Hygiene overlap; we meet on a common platform, we each have our clearly defined sphere in the matter, and it is certain that meat and milk inspections can only be efficiently performed where the two professions combine in the whole work of guarding the public health.

It would be a false position for the Veterinary Hygienist to state that such and such food was fit for consumption; he can say in the case of meat that the appearance was that of perfect health, or that the change, if any, had caused no important alteration in the tissues; with the medical officer alone must rest the responsibility of rejecting or passing it; the same argument applies to milk; we can say that the animal presents no sign of disease, that local or other ailments are in our opinion of an unimportant nature; obviously, we cannot decide as to whether the milk is fit for food, any more than the medical officer can assert that the local conditions we regard as trifling are to be taken in a serious light.

The last branch of Special Veterinary Hygiene, to which I will only briefly allude at the present moment, is the production of animal lymph for the purpose of human vaccinations; here, again, we meet the medical officer on a common platform. In the abstract, we know nothing of vaccination as a protection against small-pox, but as Veterinary Surgeons we do know cow-pox; we understand the best conditions under which it can be cultivated, know exactly at what period the most suitable lymph for the human subject can be removed, and have a clear comprehension of the feeding, management, and handling of the animals employed in its cultivation.

Briefly, therefore, these are the aims and scope of Veterinary Hygiene; we seek to make ourselves of use to the public in many varied capacities; we can advise the breeder on the selection of stock, the Government on the soundness of its horses, and on their management and care; the large and small horse owner on the feeding and general management of his stud; the farmer on the prevention of disease amongst

his stock; the State on the best methods of excluding and combating epizootic disease; and the medical profession we can enlighten on those diseases transmissible from animals to man, the condition of meat and milk from a veterinary point of view, and can supply them with a never-failing and safe source of animal lymph; we may claim, therefore, that we have a distinct sphere of usefulness; how far we are prepared at present to occupy this sphere is a question I proposed to deal with later on.

It is a remarkable thing that people who, so far as they themselves are concerned, will take every reasonable precaution to preserve their health, forget to or do not see the necessity of applying the same laws to animals which are their property. I need not remind you that there is one Hygiene in the same way as there is but one Pathology. The laws which regulate the health and welfare of the human race, equally apply to the animal community, with certain modifications which do not affect the correctness of the statement; we have, therefore, every reason for saying that the magnificent results obtained by human hygienists may also be obtained by veterinary, and more than this, the latter are capable of showing the value of their sanitary reforms in actual money. The introduction of sanitary science into the French army saved 25,000*l.* per annum in the purchase of horses alone; the invasion of cattle plague into this country cost 5,000,000*l.* sterling.

Owing to the absence of statistical data, it is impossible to assess the value of sanitary science in our army, but we do know that diseases which a few years ago were common are now never seen, or if cases occur, they can clearly be traced to outside sources of infection; the result of this is that we have prolonged the useful life of army horses, and placed them under such favourable hygienic conditions that no European army can in this respect compare with ours. The saving of money must therefore be considerable.

No matter how perfect the machinery for the suppression of epizootic diseases may be, we cannot hope to successfully cope with outbreaks without the intelligent co-operation of the public; our sanitary triumphs in the army have been won simply by the assistance rendered by higher authority; it is here that we fail in civil life, every individual considers he has no right to be interfered with, or that his liberty of action must not be unduly controlled. One of the most recent examples of this prejudice is the considerable ill-feeling with reference to the muzzling order for the prevention of rabies. So unnatural is the selfishness of the dog-owning public, that the misery and horrible death inflicted upon their fellow creatures is a trifling matter compared with the inconvenience occasioned to the canine pet by having his head enclosed in a wire cage.

It is certain that if the causes operating in the production of disease were better known to the public, their appreciation of sanitary laws for the suppression of such would be correspondingly increased, and this is one of the useful results which we may expect to follow as the result of this Congress.

At no time in the history of medicine has more attention been directed to diseases transmissible from animals to man; it is not too much to say

that some of these are positively under the control of hygiene, of which the best example is glanders of the horse; as to others we are not so clear about the causes influencing their production, but one thing is absolutely indisputable, and it is that where epizootics break out amongst animals living under sound sanitary conditions, the mortality is low, whereas, on the other hand, when such diseases appear in animals living under mal-hygienic conditions, the mortality is high, and convalescence protracted; the best example of this statement is influenza.

A consideration, therefore, of the important factors operating in the spread or production of disease amongst animals must now occupy us, and I will first deal with the questions of *Water*.

The generally accepted notion is that any water is good enough for animals, and certain arguments are used to prove this, one being that horses prefer dirty water from a pond to clear well-water. The argument is not, however, sound; the explanation is that he prefers soft to hard water. There can be no doubt that, in order to maintain a perfect state of health, a pure water-supply is absolutely necessary for animals, but little attention has been given in this respect, especially to cattle. We are aware that there are forms of organic matter in water which appear to produce no ill effects on animals, probably for the reason that they are not susceptible to any fever resembling typhoid, but sewage in water without doubt affects them, producing abortion in pregnant animals, malignant and other forms of sore throat in cattle. I have never, however, witnessed any ill effects in the horse, probably for the reason that the amount partaken of was insufficient to produce harm. Animals have been known to suffer from diarrhœa when partaking of water from a well within 100 yards of where diseased animals had been buried the previous year. The flesh and milk of animals receiving a polluted or foul water-supply has often a bad taste and odour.

Sulphurous acid finds its way into water in certain manufacturing districts; its effects on animals is to give rise to diseases of the bones. Water charged with lime has been known to produce bony enlargements in horses, and in the form of sulphate and carbonate of lime it produces certain changes in the digestive canal, resulting in loss of condition and indigestion.

Goitre had been known to affect horses and mules in those districts in France where goitre is common in man, and is attributed to the nature of the water-supply. Cystic calculi, particularly in sheep, have been attributed to hardness of the water; this disease is certainly more common in the limestone districts.

Water is well recognised as a means of transmitting certain epizootics. The London water-trough is perhaps the most common means of transmitting glanders in horses. Foot and mouth disease, cattle plague, and anthrax, are all readily transmitted by the water.

The points we would urge with regard to water are these:—

More care in obtaining a pure source of supply and in preventing it from becoming polluted.

Soft water, if possible, to be given; stagnant ponds avoided, those ponds containing living vegetable material being probably

much safer, owing to the oxidising power of growing vegetable life.

The protection of wells, ponds, and streams used for cattle and horses from sewage and other pollution.

Cleanliness of water-troughs, buckets, and other vessels used for watering from.

An unlimited supply; horses are often stinted.

The greatest care is required in the watering of horses; owing to their anatomical construction, one of the golden rules of feeding should be that horses are to be watered first and fed afterwards; neglect of this simple hygienic law entails a heavy loss of life annually, the cause of death being rupture of the stomach or intestines, or twist of the latter, all resulting from an attack of ordinary colic.

The *Air* of buildings inhabited by animals has received very little care or attention excepting in the army; here for many years past no money has been spared to render the atmosphere of stables pure and free from smell, and our efforts in this direction have met with remarkable success.

It is difficult enough to make a man believe that he is risking the health of his animals by feeding them on mouldy food, but when he is told that he is risking their lives by making them breathe foul air he positively declines to believe it, his argument being that the animals must be kept warm. The first efforts in veterinary hygiene were directed against this pernicious practice of closing doors and windows and practically suffocating animals. We have to remember that any warmth in a stable or cowshed not heated artificially practically arises from the heated air from the lungs of the occupants; in veterinary hygiene, then, heated air may always be regarded as foul air, for not only is it charged with the impurities from the lungs, but also from the decomposition of excreta. The carbonic acid in the air of stables is, with care, a perfect index to the respiratory purity of the atmosphere. The care which, in the case of stables, has to be exercised in selecting a sample of air for the determination of $CO_2$, is to take it at least 6 feet from the ground, otherwise the presence of ammonia arising from the decomposition of urea affects the analysis by Pettenkofer's method.

From over 250 analyses of stable air I obtained some interesting results. 1. The purity of the air depended upon the type of stable; where ridge or louvre ventilation existed in the roof the air was comparatively pure, the changes being frequent. 2. In those stables which had dwelling rooms and lofts over them, the atmosphere was generally foul, assisted also by the fact that in this class of stable windows are few and far between.

The above experiments showed that Professor de Chaumont's number for permissible organic impurity, viz., 0·2 of $CO_2$ per 1,000 vols of air, holds good for stables. During this inquiry some very considerable quantities of $CO_2$ were found, 2·65 per 1,000 being the largest.

Leblanc found in a stable in the Ecole Militaire 0·7 per cent. $CO_2$, and 20·39 per cent. O. Such impurity as this I have never met with.

Ammonia is largely met with in stable air, arising from the decomposition of urea.

The diseases produced, aggravated, or spread by impure air are very numerous. We shall have to show, under the head of dietetics, that the majority of preventible diseases amongst animals are traceable to food and feeding, but certainly next to this comes impure air. There is no error which gives us greater trouble to rectify in the public mind than the common one that all windows and doors must be shut if horses are to keep in health; to such an extent is this carried, that not only are doors and windows closed, but every other means of inlet and outlet, and in days gone by even the keyhole was not forgotten. The state of this atmosphere defies description; the pungent ammoniacal vapours bring tears to the eyes, the insufferable odour produces a feeling of nausea and faintness, and in such places as these horses and cattle languish. Is it any wonder that glanders and farcy are so common in all our big cities, that pneumonia is so fatal, and blood diseases such as purpura only too frequent. The production of these is aggravated by the unhealthy condition of the atmosphere in which these animals exist. Before ventilation of stables was established in the army through the intervention of Professor Coleman, we have it placed on record that hundreds of animals died yearly from chest diseases, glanders and farcy; by the introduction of a proper system of ventilation this in course of time was completely stamped out, so much so that many of us have not seen a case of glanders for years, and though we have no entirely lost sight of pneumonia, yet cases are not so severe as they were, and are consequently more amenable to treatment. It is impossible to deny that these results have been obtained through thorough ventilation and good stable management.

Much yet requires to be done in this direction in civil life, the prejudice to be overcome is immense; buildings for horses and cattle in big towns and cities in particular require that a thorough system of ventilation should be instituted, and we can only hope to establish this by instructing the public in the laws of health.

If it be possible to erase from the Government returns such diseases as glanders, it is equally possible in civil life. I have alluded to the mortality among the French cavalry horses previous to sanitary science being adopted by them. The admissions for glanders fell from 23·32 per 1,000 in 1847–52, to 7·24 per 1,000 in 1862–66, and during the same period non-specific diseases of the lungs and pleura fell from 104·7 per 1,000 to 3·59 per 1,000. The only conditions present to account for this remarkable change were increased facilities for ventilation and larger cubic space.

Coleman, who did for veterinary hygiene at the early part of this century what Chadwick and Parkes did for human hygiene some years later, held the view that glanders could be generated by the foul atmosphere of stables, and he quoted, amongst other examples, the fact that if horses on board a ship were battened down during heavy weather,

glanders occurred amongst them in consequence. It was this view of the poisoned atmosphere of the stable which laid the foundation of veterinary hygiene in this country. Whether the view is tenable or untenable in the present light of science this is not a fit place to discuss, but it is an excellent working hypothesis, and has been the means of ridding the army of glanders.

Another disease which has nearly disappeared is specific ophthalmia of the horse, a disease which has no prototype in the human subject, but which steadily though surely leads, by repeated attacks, to entire loss of vision. Foul atmosphere and putrid ammoniacal vapours were blamed as the cause of it, and the disease has certainly become so rare since ventilation and drainage were adopted, that in the army a case is very rarely seen.

A disease, called in France stable fever (analogous to hospital and prison fever in man) existed in stables abroad as the result of a foul atmosphere, and in such places wounds rapidly became putrid and produced pyæmia.

Apart from diseases of a specific kind, animals kept in badly ventilated buildings are very susceptible of cold, they require more food for the amount of work performed, and colds and coughs are frequent amongst them.

A consideration of air leads us up to the important question of *Ventilation.* This is principally carried out by natural means, though there is no reason why, say in the case of milch cows (where too free natural ventilation would unduly lessen the temperature), artificial ventilation should not be adopted; in fact, it would probably be required in winter time, if the animals were to receive the amount of fresh air they needed.

The points we have to bear in mind are these:—1. It is a simple matter to ventilate a building with an open roof, viz., one with no rooms or loft overhead, but where closed roof stables exist with but few windows, and those often badly arranged, ventilation becomes nearly impossible. 2. No stable or building for animals can be effectively ventilated by natural means where the width of the building exceeds 30 to 35 feet. Windows opposite to each other should exist on both sides of the building, between these windows not more than two animals should be placed. The old idea of having windows only at one side of the stable or only at the ends of the building, with perhaps eight, ten, or more animals between opposite sources of air is the worst method which could be devised.

*Food.*—The complicated apparatus essential to the digestion of vegetable food exposes the possessor to a very large number of diseases unknown to omnivora. Besides those disorders attributable directly to food and feeding, we have many others only attributable indirectly to this cause, and yet the sum of these presents an astonishing total.

It is when we come to consider food and feeding as a branch of hygiene that for the first and only time we cannot lay down one set of laws applicable to all animals, for the reason that each class is fed

on a different principle, due to the arrangement of the viscera. Nothing can be more different than the feeding of horses and cattle, or sheep and pigs, yet all these animals, by means of their wonderful digestive laboratory, are capable of converting vegetable into animal tissue. To give some idea of how vastly these animals differ in the arrangement of their digestive tract, I may say that of every 100 parts of gastro-intestinal canal, the stomach in the horse is represented by 8 parts, in the ox by 70 parts, in the sheep by 66, and in the pig by 29 parts.

Animals are fed either for work or for food. In either case the principle of giving sufficient to supply daily wear and tear is obvious; horses cannot work, nor cattle fatten, nor sheep thrive on a poor or illiberal diet.

There is, perhaps, no branch of hygiene more under our control than food and feeding; the public know very little about it, and so many preventible diseases are attributable to this cause that it is clear some elementary instruction should be given to horse and stock owners.

The diseases are due to either alterations in quantity, quality, or conditions of digestibility and assimilation.

Amongst the wealthy class of horse owners the tendency is to cram animals with food, leading to deposits of fat on the body, often rendering even light work a matter of considerable difficulty; this is particularly the case with carriage horses. With hacks and hunters the matter is different, the amount of work performed being equal to the amount of food given, very little is stored up in the system. An excess of food leads to diarrhœa and other disturbances, the undigested food undergoing certain changes in the bowels probably with the development of leucomaines, and not unfrequently giving rise to liver disturbance as well as bowel irritation.

A large quantity of nutriment being poured into the blood leads to changes in that fluid, especially if the animal has for some time previously been on a poor and innutritious diet. Symptomatic anthrax in cattle is attributed to this cause in England, and in India the fattest horses are invariable the victims of the virulent anthrax of that country. Excess of food leads to liver changes, inflammation of the lymphatic vessels of the limbs in horses, paralysis of the stomach, gastric apoplexy, colic, and inflammation in the feet. In cattle, gorging of the stomach, tympany, impaction of the stomach, and other disorders are directly attributed to excess of food, particularly of a special kind, tympany, for instance, to excess of green food, the same cause producing tympany in the horse.

With a deficiency in food, certain marked physical changes occur in the organism accompanied by diseases which we regard as due to neglect. Scabies, glanders, farcy, and dysentery occur in horses exposed to privation and severe work, such, for instance, as occurs to animals in war. Hæmoalbuminuria and Moor-ill in cattle are attributed to a deficiency in food. With a deficiency in saline matter, rickets and other diseases of nutrition follow; with a deficiency in albuminoids there is loss of energy, and with a decrease in digestible and an increase in indi-

gestible matter there is a general loss of muscular development and pendulous abdomen. We have before noted the importance of very gradually bringing animals which have been living on comparatively poor food on to diet containing more nourishment than that which they have been used to.

Diseases associated with the digestion and assimilation of food are exceedingly numerous. There are some grains which if given to animals act like poisons. Wheat, for example, will produce tympany in horses, cattle, and sheep; maize has been blamed for producing bowel and skin diseases, and in the horse laminitis; softening of the liver of horses in Egypt has been said to be due to a kind of clover. Potatoes purge horses, buckwheat has been said to produce cerebral derangement, and parsnips to affect the eyes. Laryngeal paralysis in horses has been traced to the use of Indian vetches, and acorns have been known, in certain years, to be exceedingly fatal to pigs, probably owing to their astringency. Eczematous skin disease, common amongst horses in India, is due to feeding on wet grass.

Conditions affecting the quality of food is a very important question to the veterinary hygienist; it embraces the growth and preservation of the plant, the nature of the soil on which it is grown, and a variety of animal and vegetable parasites which infect it.

The quality of the soil materially affects the nutritive properties of the grass and grain grown on it; the time of preservation and the nature of the weather influences the feeding-value of hay and grains; the weather also seriously affects the preservation of food, wet and damp inducing the growth of vegetable parasites, which not only render the food useless as forage, but in some cases actually poisonous. In this connexion, we consider bunt, rust, mildew, smut, and ergot, which affect the plant during its life, and the mucors and penicillium which generally affect it after its death; we have no direct evidence as to the invariable harm produced in the system by these parasites, though we are all agreed that they are prejudicial to animals, and that in many cases they may produce diabetes, nephritis, paralysis, inflammation of the bowels, and cerebral symptoms.

The quality of food is also affected by its age; new hay produces diarrhœa and is innutritious; new oats cause purging and stomach irritation. By keeping, these harmful properties are got rid of.

Cleanliness of food is an important condition of quality; dirt finding its way into the intestinal canal is liable to accumulate and produce considerable irritation and death. Calculi have also been supposed to be produced by the same cause, though it is possible that in these cases their only effect is to act as nuclei for the deposition of the mineral matter in the food.

The preparation of food affects it digestibility. Old horses with defective teeth need their corn to be crushed, and corn crushing, even for horses with perfect teeth, facilitates digestion; cattle should have their roots sliced or pulped to prevent choking; maize and barley, beans and peas should be crushed; some grains used for feeding in India require to be boiled, and boiled foods are often used for fattening horses in this country.

Boiling food is a serious error, and fraught with danger to horses; it is a prolific source of stomach and intestinal disorder, partly, perhaps, from its soft nature not producing stomachic peristalsis, but largely from the fact that boiled foods are "bolted" without being masticated.

Food is also affected by the presence in it of poisonous plants, and cattle and sheep poisoning from this cause is common, but we have said enough to show the importance of care and attention to food and feeding, and the enormous influence this presents in the preservation of health. When an outbreak of disease of a non-specific character occurs amongst animals, the food, its quality, nature, &c., is naturally, and must ever be, the first question we inquire into.

*Stables.*—The large majority of buildings existing in towns and cities as stables and cow-houses, &c., are not worthy of the name. The London cab stable is typical of the class, low, dark, oppressively close and muggy, flooring bad, no drainage, atmosphere reeking with ammoniacal vapours, which to a new comer irritate the conjunctiva beyond conception; the whole place tumbling to pieces, doors patched up with sacking, windows in a similar state, or filled in with straw, soiled and foul bedding lying everywhere, and the level of the stable floor not uncommonly below the level of the surrounding soil. Amongst the better classes we gradually find these structural defects remedied, but the absence of doors and windows in sufficient number for lighting and ventilating purposes is very marked. It is only amongst some large companies and in the army, that stables constructed on a sanitary plan exist, and to these we shall presently refer.

Cow-sheds in cities and towns are much more under control than stables; the watchful eye of the medical officer of health ensures at least the elements of sanitation being observed, but much yet remains to be done in construction, lighting, flooring, ventilation, drainage, and heating, before we can consider that milch cows are placed under healthy conditions.

The principles of construction are very clear: use virgin soil where possible, avoid made ground, low lying places, or soil in which the ground-water is high. It is not much use suggesting a deep gravel where such is not the natural geological formation, but such a site is preferable to any other.

Let the walls be double to ensure dryness, the foundation and flooring of concrete to avoid the permeation of urine and fluid fæces, the width of the building not to exceed 30 feet, the animals to be arranged with their heads to the outside walls, with a passage down the centre; a large Sherringham window over the head of each animal, doors at either end, the roof open, *i.e.*, no rooms overhead, with louvre ventilation on the ridge. The fittings to be of iron, plain and strong, the brick flooring which is placed over the concrete to be well set, not slippery, durable, impervious, readily cleaned; the drainage everywhere inside the building to be surface gutters, a very shallow one from each standing running into a wider one which runs at the rear of the stalls the whole length of the stable. Ample water-supply for flushing stalls, boxes, and drains; no traps or gullies inside the building, and the walls, as high as the animal

can reach, to be of cement. The cubic capacity per head should be 1,600 feet, and the superficial area of the stall 70 feet, and with share of central passage, 100 feet. Such are the principles on which the most approved type of stable should be built.

The objection to the cubic contents named is that the building would be cold; where this objection holds good there is no reason why artificial warming should not be practised by means of a gas bracket at certain inlets, the products of combustion being carried off from the building, or hot water pipes may be used. The present practice of raising the temperature of the building by the heat radiated from the body and by the heated expired air is obviously highly insanitary.

The surroundings of the stable or cow-house need attention: dung-pits, uncleansed drains, &c., should be looked to; pits intended for manure should not be nearer than 13 yards to a stable. How often do we not find them placed close to or under the window, the poisonous vapour passing into the building? Care should also be observed not to crowd buildings too close together, and the nearest that one block should approach another is twice the height of the nearest building. Blocks built in the form of a square are bad, especially if close together, the obstruction to free natural ventilation being very great.

In dairies, special provision must be made for rooms for storing the milk, which should be quite distant from the cow-shed or any human habitation.

All drains should be properly trapped, and if sub-soil drainage is introduced into the building all traps should be ventilated, and thus cut off from the sewer.

It may not be possible, owing to the expense of construction and other causes, for private individuals to afford their animals such sanitary dwellings as we have described, but an endeavour should be made to work as near to it as possible.

All receptacles for excreta should be cemented, and the same remark applies to cow-houses for the place where "grains" are stored.

The removal of excreta is an important matter: we are liable to forget that one of the chief causes of air impurity in stables and similar buildings is owing to the presence of fæces and urine, and if for no other reason than this ample ventilation is required. By the use of surface drains the most effectual and sanitary drainage inside the stable is accomplished, and none other should ever be used.

The part played by *Soils* is the production of disease is far from clear. We do certainly know that a high ground-water is dangerous to health, that marshes are prolific of sheep rot and anthrax, and that alluvial soils, with dense marls and clays are, especially in the tropics, notoriously unhealthy; but how far such diseases as tuberculosis, glanders, &c., are affected by a polluted soil, we have at present no information. My own impression is that in course of time it will be shown that soils do play a very important part in the production of disease; the whole question needs to be thoroughly investigated, especially in the tropics.

The nature, character, and conditions under which animals perform *Work* is a question which offers the fullest possible scope for hygienic

interference. In this country, horses are the chief animals used for the purpose of work, but abroad, and with armies in the field, mules, bullocks, elephants, camels, &c., are severally employed, and require considerable attention.

The nature of the work comprises riding, draught, and pack. In the former, the fitting of saddlery and the prevention of sore backs is a subject on which much might be written; we must here only content ourselves by saying that the question, until recently, has never been scientifically studied, the result being the production of immense injury and loss. We now know that saddle fitting can only be properly carried out when anatomy and the laws of physiology are brought to bear, in showing what part of the back is capable of bearing weight and what parts are not, and in demonstrating the absolute necessity of the saddle conforming to the shape of the horse, and the advantage of distributing the weight over a large rather than a small surface.

The ultimate result of employing these simple rules will mean, to the army in particular, an immense saving of life and suffering in the future. With pack animals, much the same considerations in saddle fitting apply as to riding horses.

With draught, a great deal needs yet be done; the best form of vehicle suited to different roads, size of wheels, angle of traction, fitting of collars and other portions of harness, and the relative advantage of shaft versus pole draught, are all points influencing considerably not only the value of the work performed, but the comfort and health of the animals called upon to perform it.

The streets of every big city demonstrate daily the necessity of inquiring into this question of draught; horses are compelled to perform their work under unnatural conditions—defective shoeing, slippery roads, and other factors which tend to exhaust their strength and wear them out long before their time.

The regulation of the amount of work to be performed can only be observed where the public is instructed and capable of understanding the elementary fact that a horse is not a machine, and that his muscles need rest and time for repair as well as our own. It is, perhaps, on points of this kind that hygiene may have the least influence; cupidity is strong. Fortunately we are a horse loving race, and the feelings of the noble animal appear to directly appeal to our senses; but in other countries such does not appear to be the case. The operations of the law give horses a certain amount of protection in respect of the question of overwork, but it is probable that in cities the major part of this cruelty is inflicted at night time.

### Individual Hygiene.

Many points of the greatest importance have to be considered under this head, such as stable management, bedding, grooming, clothing, clipping, shoeing, etc.

A good bed for horses is essential; if they lie warm and comfortable it is equivalent to extra food, the limbs are rested, and the horse

refreshed. The bedding should receive careful attention in the morning, the whole of it being removed from the stable and the soiled portions replaced, the portion of litter kept should be dried in the sun if possible, but under all circumstances thoroughly aired and ventilated.

The common practice of not removing the bedding from the stable, or of turning it under the manger during the day, cannot be too strongly condemned: it is impossible under these circumstances to keep the air of the stable sweet, owing to the amount of ammonia and foul vapour given off from the decomposing litter.

It is considered by some that horses should be bedded down during the day, as it saves the feet; certainly horses like bedding under their feet, but I am not prepared to say that such is essential.

The use of moss-litter and sawdust as bedding, with the object of absorbing the urine, is very good if the absorbent material be kept clean; where soiled bedding is allowed to remain the feet are destroyed by being soaked in the alkaline mass produced by the decomposing urine.

In *Grooming*, two objects are aimed at, viz., a clean skin and a glossy coat. A brush suffices for the former; the natural sebaceous secretion which gives the latter is excited by wisping. Grooming is an art which can only be learned by long training; its theory is simple, its application difficult. Washing horses is a pernicious and dangerous practice, dangerous for the reason that they are always left to dry themselves, pernicious because it removes the natural grease from the skin, induces cold, and causes the coat to become dull. A horse covered with dirt should be scraped, the body dried and clothed; if not wet, but simply muddy, the latter should be allowed to dry and then brushed out. Washing legs in winter produces intense erythema of the heels, sometimes reaching as high as the knees and hocks; it is due to using hot water and not drying the parts.

The skin of the limbs also suffers from the application of salt to the roads in times of snow, the low temperature produced by snow and ice causing the parts to become frozen.

Thus we find the skin suffers from want of grooming, and the limbs from washing and other causes; the amount of damage the latter produces in large cities in the winter can hardly be imagined. Scabies shows itself through neglect in body cleanliness; the disease in the horse spreads with surprising rapidity, and nothing but prompt action can effectually arrest it. I am not clear how far dirt alone produces the disease, or rather affords a nidus for its existence. I have seen bodies of horses systematically neglected in respect of grooming keep free from scabies, and I have known it occur where the best stable management existed; in this case the disease was imported. When, however, we combine dirt with poverty and starvation, at once the disease breaks out in its most virulent form, and in this state is the scourge of armies in the field.

It is a recognised fact that horses living constantly in the open require very little grooming, as it exposes them to cold and skin irritation. The surface of the body should simply be wisped, but both main and tail require thorough brushing out.

The amount of clothing required by horses depends entirely on the length of the coat and the ventilation of the stable. In cold stables they require more food to keep them in condition, and extra clothing saves this to some extent. On the other hand, the wearing of three or four blankets in the winter is a mistake, as the coat grows thin and fine, and the sudden change from, perhaps, a hot stable to a piercing easterly wind, is the explanation of most of our cases of chest disease. Army horses receive no clothing, no matter how severe the winter, unless they are living in the open. This should be sufficient evidence on a large scale of the comparative immunity of the horse against cold.

*Clipping.*—The extra growth of hair during the winter causes, with hard worked horses, continual sweating during work, and in this state more harm is done than it is possible to conceive. Horses with wet skins are left standing in the streets, instead of being quietly walked about, and to this a vary large proportion of our cases of pneumonia and pleurisy are attributable. We recognise as a practical fact that if horses are performing severe work in the winter the long coat must be removed, and artificial covering take its place in the stable.

There are certain skin diseases of the legs attributable to long hair, affecting especially the heavier or common breeds. Long hair on the limbs acts as a nidus for dirt, as being coarse and stiff it is difficult to clean. This, no doubt, is one of the explanations of "grease" in the horse. The abundance of hair grown on the limbs, and so much prized by breeders, is a source of danger, and is no indication of strength as is commonly supposed.

The *Shoeing* of horses is a most important practical side of veterinary hygiene. Though the most intelligent horse owning race in the world, we are, nevertheless, sadly behind in understanding the principles of an art which contributes so largely to the soundness and comfort of horses.

We cannot here enter into the unprofitable discussion of whether horses could or could not work without shoes, but must at once recognise the practical fact that shoeing is a necessary evil, like many other things the outcome of the domestication of the horse. The object of shoeing is to prevent undue wear of the foot; this protection is given by a rim of iron nailed to the part. In applying this we must bear in mind the physiology of the foot, so as not to interfere with its functions more than we can help, and as nails have to be driven into living structure, we must by care reduce the evil thus produced to the narrowest limit. The function of the wall of the foot is to bear the weight of the body; the same remark applies to the bars; the use of the sole is to protect the delicate parts inside, and, by its union with the wall, to assist in supporting the weight. The function of the frog is to prevent the horse from slipping; by its physical character, viz., india-rubber nature, it prevents concussion to the foot and limb, and through its position it supports weight, keeps the heels open, and so maintains the proper width of the foot. The elasticity of horn is obtained by the amount of moisture found in its horn tubes; when these are destroyed the moisture

evaporates, and the part becomes dry and brittle. Brittleness occurs when the wall is rasped, as is commonly done in shoeing; it is equivalent to removing the enamel from our nails by scraping.

This bare outline of the physiology of the foot tells us that the shoe should fit the foot, and not the foot the shoe, that it should have a perfectly level bearing on the wall and bars, that the sole should not be touched with the knife, that the functions of the frog are entirely destroyed by allowing the part to be cut away, and that it can only act and grow healthy and strong by resting on the ground. Physiology also tells us that no more nails than are necessary should be placed in the foot, and that the wall should not be rasped on its surface.

The function of the foot does not teach us the pattern of shoe needed. This depends upon the nature of the work, but the simpler in form the better.

So impressed is the public by the necessity of having different shapes and makes of shoes, that they have lost sight entirely of the infinitely more important point—the fitting of the shoe. My own opinion is that, providing the shoe conforms with the principles of shoeing here laid down, the pattern of shoe used is immaterial.

By care and attention to shoeing thousands of pounds worth of property would be saved to the country yearly. There are no greater banes of horseflesh existing at the present time than the incompetent farrier and the ignorant owner. The latter is solely to blame; he leaves the shoeing of his horses in the hands of ignorant and prejudiced servants, and the farrier has to do as directed by them or lose the work. The principles of shoeing can be learned by a child, and I look forward hopefully to the not very distant future when our English horses will be shod on scientific principles, and this only needs the hearty and intelligent co-operation of the educated public.

## State Hygiene.

So far I have only attempted a brief consideration of the important factors in the production of diseases which we have shown are largely under our control, and almost entirely in our own hands. I must now draw attention to the existence of certain diseases which are not so clearly controllable, are capable of inflicting enormous loss, and require prompt and energetic action to combat them. I allude to the many epizootic diseases affecting horses, cattle, sheep, and pigs, in this country.

There are certian epizootic diseases affecting all animals; there are others affecting only certain members of the same class, and others again which only affect animals of one kind.

Anthrax is a disease affecting nearly all animals, and equally fatal in each. Variola is another affecting nearly the whole animal kingdom, but with different degrees of severity, being exceedingly serious and fatal in the sheep, benign in the horse and cow. Rabies affects all, and is fatal to all. Tuberculosis, common in cattle, rarely attacks sheep, and still more rarely horses. Specific pneumo-enteritis is essentially a disease of

the pig, known as swine plague. Cattle plague and pleuro-pneumonia affect horned cattle only. Glanders affects only solipeds, and man through inoculation. Foot-and-mouth disease attacks cattle, sheep, goats, and pigs. Foot-rot and liver-rot affect only sheep. Influenza and strangles are essentially diseases of the horse.

As a matter of fact, it may be stated that none of these diseases need really exist; most of them can be completely stamped out. Some, owing to the vitality of the poison, are difficult to eradicate, of which the best example is anthrax; but assuming, as we are bound to, that of the purely specific diseases mentioned above none can arise *sui generis*, then the only logical conclusion is that stamping out should rid us of them for ever.

It would be difficult to imagine a country more perfectly suited to the exclusion of disease from foreign sources than the British Islands; it is true that our requirements in foreign cattle are large, and that we draw from notoriously infected countries, or at any rate, from countries which have been affected; still, given the conditions essential to prevent the introduction and spread of epizootic disease, we find that in this kingdom they exactly exist.

When diseases of a contagious or infectious nature affect animals, there is no reason why, so long as we know the definite conditions under which the poison is spread, that we should not be able to arrest its progress. We have this information for such diseases as anthrax, cattle plague, pleuro-pneumonia, foot-and-mouth disease, variola, foot-rot, glanders-farcy, rabies, tuberculosis. We have not got this information in respect of influenza or strangles. I do not mean it to be understood that the profession is unanimous in its views on these points, but we are pretty clearly agreed that glanders can only affect by contact with the poison, and that simply living under the same roof with a diseased horse will not infect a healthy one which has not come into contact with the diseased patient; and the same remark applies to rabies, anthrax, foot-rot, and foot-and-mouth. These diseases, like mange, require for their propagation actual contact with the poison, though not necessarily with the diseased patient.

Cattle plague, pleuro-pneumonia, variola, and probably tuberculosis, are undoubtedly diseases spread through the medium of the air of infected areas. Healthy animals introduced into sheds occupied by the sick, or into places where affected ones still exist, may contract the disease, though no actual contact has occurred.

Influenza and, perhaps, strangles, affects horses quite irrespective of any condition; contact or no contact, infected buildings or otherwise; we have certainly reason for believing that the poison in the former disease may be carried for long distances by the air, and that all susceptible animals are affected by it.

There are thus three distinct classes of epizootic disease, and the efforts control of these increases in difficulty from I. to III. Class I. can be absolutely stamped out, as we know exactly the lines on which to limit the spread of the poison, which is a fixed one. Class II. is more difficult, and apparently even slight air-infection is sufficient to produce

the disease; the poison is both fixed and volatile. In Class III. the poison is probably wholly volatile, and is absolutely not under human control.

The methods to be adopted to rid ourselves of disease will depend in the first instance, as to whether, like foot-rot, foot-and-mouth disease, mange, influenza, strangles, &c., the disease is distinctly curable (*a*); or whether, like anthrax and cattle plague, it presents a high mortality (*b*); or like glanders, rabies, pleuro-pneumonia, and tuberculosis, it is absolutely incurable (*c*); for practical purposes we may class *b* and *c* together, and advise destruction; the number of recoveries in class *b* is small, and does not counterbalance the increased risk attending the keeping alive of such foci of infection.

I have previously stated that theoretically we should be free from animal scourges; the advantages we have over the human hygienist in this respect are enormous, we can destroy the affected, and segregate the sound or exposed. Nothing remains to us but the destruction of all affected in class *b* and *c*, and to a great extent the destruction of all absolutely in contact with the infected; this destruction must include not only taking away the life of the animal, but the destruction of the body, so as once and for all to pevent its producing further harm. Perhaps it is in this latter respect we fail more than any other; we allow, it is true under certain conditions, the utilization of hides, hoofs, horns, and even flesh from the infected, and thus most considerably contribute to the support of the life of the disease-producing organism. Occision should include destruction in its fullest sense. Isolation is the method employed for stamping out the diseases in class *a*, and for completing the measures adopted in classes *b* and *c*.

This isolation must be of the most perfect kind, such as can only be obtained by the assistance of the law; infected places should practically be in a state of siege; though it is clear that such rigorous measures are only necessary when the diseases are such as are undoubtedly conveyed by the movements of animals and by the attendants on the sick, they must be judged of also by the intensity of the poison.

Depending, again, on the nature of the poison, must such measures as the movements of all animals in the district, holding of fairs, &c., be prohibited; such measures, though extremely productive of public inconvenience are essential in such diseases as sheep-pox, pleuro-pneumonia, swine plague, glanders, &c., &c.

The theoretical exclusion of disease from this country is obtained by means of our system of port inspection. I am not prepared to say that this latter is not capable of further improvement and extension, but I do say that this enormous work so quietly and unostentatiously carried out is the salvation of our herds; it is impossible to estimate the value of our port inspection; that certain cases must escape detection is inevitable where so many thousands are being inspected, and when we consider the obscure symptoms of some of our most contagious diseases, notably tuberculosis and pleuro-pneumonia.

The value of quarantine is also fully appreciated where stock is landed from suspected countries, and the necessity of destruction at the

port of debarkation, though full of inconvenience to the public, is a method of prevention of the greatest possible utility.

The laws regulating the transport of cattle across the country by rail and road appear to meet all requirements; they are not intended to apply to diseased stock, though of necessity, such cases must leak in. The disinfection of railway trucks is an important matter. I am rather inclined to regard the lime-washing as a useless measure when animals labouring under specific diseases have been carried; from a point of cleanliness, it is of course useful. I consider that railway trucks should be disinfected by means of super-heated steam.

The following table, compiled from the Reports of the Agricultural Department, will give some idea of the losses arising from contagious and preventible diseases of animals in Great Britain:—

**Number of Cases of Contagious Diseases of Animals in Great Britain.**

Compiled from the Agricultural Department Reports.

| | | | |
|---|---|---|---|
| Cattle plague (21 years) | 88 | Sheep-pox (none for 21 years) | — |
| No cases since 1877. | | Sheep scabies (21 years) | 883,933 |
| Foot-and-mouth disease (24 years) | 1,853,145 | Swine fever (21 years) | 272,578 |
| No cases since 1886. | | Glanders (21 years) | 14,931 |
| Anthrax (4 years) | 1,142 | Farcy (17 years) | 5,797 |
| Pleuro-pneumonia (21 years) | 78,198 | Rabies (4 years) | 1,147 |

The transport of cattle by sea is a question which has gradually forced itself on the attention of the public. The profession for years has been alive to the cruel conditions under which this is usually carried on. Larger air and deck space, better ventilation, specially constructed pens, and greater cleanliness are required. The fittings should as far as possible be of iron to facilitate disinfection, and should be securely fixed. The only real solution of the difficulty is the special construction of ships for cattle traffic, and none others should be employed.

## Special Veterinary Hygiene

To the special branches which fall under the head of veterinary hygiene I have already previously alluded, it is here only my intention to describe one to which, perhaps, increased attention will be paid in this country. I allude to the cultivation of calf-lymph for human vaccination.

It does not fall within my province, nor is a paper of this kind is it desirable, to discuss the relative advantage of humanised and of animal lymph. For our purpose it is sufficient to assume that animal lymph is required, and we have, therefore, only to point out in what way and under what conditions this can best be obtained, and endeavour to show that lymph so cultivated produces in the human subject a typical eruption, which we have every reason to believe is as protective as humanised lymph.

Calves are selected as the animals to be worked with, partly on account of their manageable size, but also from their less liability to be infected with such diseases as tuberculosis, and moreover, because it is not improbable that the eruption is more typical, and the yield of lymph larger in the young than in the adult animal.

The most suitable age is found to be between three and six months old; they should be healthy, well nourished, and weaned. This latter is an important point in the sense of economy, the expense of feeding the animals on milk being considerable.

Every animal, before being used for cultivation purposes, should be kept under observation for some days, and during this time the temperature should be regularly taken morning and evening; this period also enables one to determine whether the diet they are placed on agrees with them, for calves are most liable to a fatal form of diarrhœa from errors in feeding, alteration in diet, and other causes unnecessary to be named. Animals affected with ringworm or eczematous looking skin should be rejected.

The most suitable daily diet for a calf of the age mentioned is, hay, 4 lbs.; grains, 2 lbs.; bran, 1 lb.; oil cake, 1 lb.; mangles, 5 lbs. I believe that on a poor diet the yield of lymph falls off, and that it is economical to feed them liberally if only on this account.

The sanitary conditions under which the animals live are very important to attend to; the surface of the body should be kept clean, and all soiled portions of the skin regularly attended to; they should be fed and watered three or four times a day; the stable in which they are kept must be clean, warm, light, and well ventilated, with surface drainage, walls regularly whitewashed and impervious; flooring dry, clean, and sweet, and the partition separating one animal from another should be whitewashed two or three times a week. The stalls erected for the calves should be sufficiently wide to allow them to lie down, but not to turn, the object being to prevent them getting at the vaccinated surface and destroying it by licking. The animals must be kept clean; to avoid them getting soiled by lying in their own fæces regular attention is required, and each calf should be allowed 4 lbs. of straw daily as bedding. The warming of the building must be carried out by hot water pipes.

The vaccination is readily performed; the animal by means of a special table is placed on its side, and the hind legs being separated, the abdomen and inside of both thighs are carefully shaved, washed, and dried. I vaccinate over a large surface but never carry the insertions below the middle line of the abdomen, as they would only get soiled and destroyed.

The number of insertions are from 30 to 50, depending on the size of the calf. There is no reaction in the system depending upon the area covered; it makes no difference, either to the state of health or to the temperature, whether we make 30 or 50 insertions.

The form of scarification is most important. I vaccinate over a square or circle, the diameter of a shilling to a halfpenny, by a series of light diagonal scratches which cross each other; no blood should be drawn; the lymph is then carefully worked in. I allow a few minutes to elapse after the last scarification before the animal is taken off the table.

There are other methods of infecting the surface, namely, by a single slight incision which only passes through the epidermis, or by punctures; both these methods in my opinion are defective, inasmuch as

they are wasteful; by the circle or square method three or four times more lymph may be obtained.

There is no rise in temperature for two days; on the third day a marked elevation generally occurs, but the health of the animal is not affected as judged of by the appetite; on the fourth day it is evident that the scarifications are taking, as indicated by a red areola round each; on the fifth day the vesicle is surrounded by a silvery areola, outside of which is a narrow red one, the vesicle is yellow in colour, the centre being depressed, darker, and drier than the surrounding parts.

On the fifth day the calf is placed again on the table, and the whole part thoroughly washed. For this purpose I use a hose attached to a tap, and every particle of soiled material is removed; by this time the peculiar characteristics of the vesicle become fully developed. It is remarkable how they come out through washing, a most unpromising looking eruption after a thorough wash turning out a typical one. The surface in being sponged should not be rubbed, or the vesicles are damaged.

It is as well to starve the calf on the morning it is required for collection, as owing to the length of time it is on the table the stomach becomes partly paralysed, fermentative gases collect, and the abdomen becomes distended. This is avoided by starvation.

Operators differ in their views as to the period to be selected for lymph collection. On the continent the sixth and even the seventh day is selected, whereas, in this country the fifth day is the one generally, and I think properly, chosen. It is certain that in a small percentage of cases, the vesicles on the fifth day are not up to time, and these may be allowed another day, but such cases are the exception.

It is necessary that we should be able to carefully discriminate between the vesicle fit for lymph collection and the one which is too far advanced to admit of its being used for vaccination.

The typical vesicle fit for lymph collection has a narrow red areola surrounding the vaccinated surface; the edge of this latter is raised, and quite silvery in appearance, the width of this narrow silvery edge being not more than one-eighth of an inch even in large vesicles; within this, the surface of the vesicle is yellow, darker, and towards the centre depressed, it is, moreover, dry, and if scraped is distinctly firm, separating with difficulty from the part below. The advanced vesicle is yellower, the red areola much larger, the silvery one disappearing, and the yellow, depressed crust, instead of being adherent, is either separating, or readily separated, exposing a red, ulcerating looking surface below, bathed in pus. Such vesicles must not be used for lymph purposes.

Assuming, therefore, that our vesicles are all that can be desired, the lymph is collected by first strangulating the vesicle with a specially prepared clamp, at first applying the clamp lightly; the surface of the part is then carefully scraped to remove crust and silver areola, and rapidly wiped; the clamp is now tightened, and, if successful, the part is rendered bloodless, or nearly so. The object of not tightening the clamp at once is to prevent forcing out the fluid lymph before the wiping process is finished. The object of lightly scraping the surface

and removing the crust is to use nothing which has been in contact with the ground or the air, and the necessity of using a clamp is to keep the lymph bloodless and to facilitate work.

Vaccine lymph consists of two distinct parts, viz., a fluid and a solid portion; some calves yield mostly fluid lymph, others yield a comparatively dry solid lymph, and in a third class the solid and fluid is equally distributed. The fluid lymph is the serum of the blood, the solid is the tissue of the pock; both are very virulent.

The first material from a vesicle is a yellow fluid, sometimes blood stained. After this is removed with a collecting spatula the next thing taken is the solid pock tissue; this is completely scraped away, and by this time the clamp will require re-tightening; the next to appear is a thin, watery, colourless fluid, more like the lymph from the human arm, it exudes from the lymphatics, and I have proved it to be virulent. It is sometimes in great abundance.

My own prejudice is in favour of the solid pock tissue, as it is easier to keep aseptic, and, I think, retains its virulence longer.

By means, then, of the collecting spatula, the whole vesicle is robbed of its fluid and solid lymph, and placed in an agate mortar. Each vesicle is treated in the same way, being unclamped after it is emptied. The crusts and silver areola belonging to each vesicle are kept separate for the vaccination of the next calf.

The whole of the lymph, which may now weigh 11 grammes, is carefully and in small portions at a time ground up in an agate mortar to a fine emulsion; by this means the fluid and solid material is intimately mixed, and anything like portions of skin can be readily removed, as they impart a lumpy feeling to the pestle which renders their detection easy and simple. After the whole lymph has been treated in this way a preserving agent is added; the best for the purpose is pure glycerine, the amount added depends on circumstances, but it is generally about equal to the bulk of lymph; the most thorough incorporation of this mixture now takes place, and it is then placed in tinted tubes $1\frac{1}{8}$ inches long by $\frac{1}{4}$ inch diameter, corks are inserted, and the whole sealed by dipping carefully in melted paraffin; this is now kept as stock, and protected from the light and heat.

When small quantities of lymph are required, say for 10 to 25 people, the necessary amount is placed between squares of glass, plain or hollow, and the edges of these are dipped in melted paraffin and so sealed; this is a rapid and most convenient method of issuing lymph, and gives the operator the least trouble. The glasses are protected from light by being folded in tinfoil. When lymph for 100 persons is required, one of the tinted tubes above described is sent, as each holds sufficient for 100 vaccinations.

Other methods of preserving and issuing lymph may be adopted, such, for example, as ivory points and capillary tubes, but I can say little for these, for the reason that the ivory point is wasteful, and some time is occupied in dissolving the lymph; moreover, the dried lymph does not maintain its vitality for much more than two months, and in

the summer not for longer than six weeks. If points are charged no glycerine is added to the lymph.

Capillary tubes may be filled direct from the vesicle; such lymph is very difficult to expel from the tube owing to coagulation, and the process is wasteful; moreover, the lymph soon gets cloudy and unfit for use. Lymph diluted with glycerine and water may readily be introduced into the tubes after the grinding process described, and this is the most successful of the tube methods, as much of the solid pock tissue also finds its way in and increases the virulence of the fluid. Personally, I unhesitatingly prefer the method of issuing between glass plates.

Calf lymph is always slightly tinged with blood. The method by which it is collected accounts for this; the tint is quite unobjectionable, and free from danger so far as we are at present aware.

There can be no doubt that part of the regular system of an animal vaccine establishment should be the destruction of the vaccinifer, and post mortem examination of the body; by this means we could completely control the issue of lymph by destroying all that derived from a diseased animal. The percentage of calves affected with tuberculosis appears, from continental experience, to be something infinitesimal; whether the disease amongst English calves is as low I am not in a position to say.

The vitality of calf lymph is an important practical question. There is no doubt that its life is a short one, and that on the human subject it begins to fall off at the second month, and at three months is almost inoperative; strange to say, however, it retains its virulence for calves for a much longer period, and I have successfully vaccinated calves with lymph 14 months old. I cannot explain this difference, but it is undoubted.

During hot weather lymph should be kept in a refrigerator, and under all circumstances it must be kept in the dark.

A very important practical point to consider is the extent to which dilution of the original lymph may be carried by means of water and glycerine, and on this point I am prepared to speak with exactitude.

The idea in this country is that the lymph should be issued on points and nothing added to it, that by adding anything an opening is allowed for septic organism, &c., and moreover the proportion of lymph to dilutent is so small that failures occur. This view is not correct; no septic organism will flourish in glycerine, and the fact is lost sight of that, the water used to dissolve the dried lymph on points may be and is teeming with organisms, which are thus readily introduced on to an abraded surface.

The vaccine organism can live for a long time in glycerine, so much so that at one time I thought it probable glycerine extracted the poison; experiments made to prove this failed, but it is certain that, where thorough incorporation has taken place, glycerine containing only a minute proportion of vaccine gives successful results.

Experiments made to determine the amount of dilution to which lymph can be exposed showed that when there was only one part of

lymph to five parts of glycerine, as good results were obtained as when lymph alone was used; even when the lymph formed but one-sixth part of the vaccinating fluid good results were obtained.

The disadvantage of highly diluted lymph is that it cannot be neatly sent between glass plates.

It is no uncommon thing after the use of calf lymph, no matter how prepared, for the arms on the eighth day after vaccination to present a highly inflamed appearance, the limb being swollen and the erythema extending as low as the elbow and as high as the axilla, the part being abruptly defined from the surrounding skin. The lymph is often blamed, and it may be responsible, but such conditions occur principally in adults, and generally in those persons not remarkable for body cleanliness; I attribute the trouble to infection from soiled linen and dirty skin, and to using instead of resting the arms; exactly the same lymph used on infants produces no such erysipelatous eruption, the explanation being that they are kept cleaner and cannot scratch the part. I have never seen any cause for anxiety in such cases, the redness disappears in a day or two, leaving some œdema which gradually spreads through the length of the limb, invading forearm, wrist, and hand.

I have seen several cases in adults where swelling and redness of the arms have occurred without a suspicion of body cleanliness being defective; such cases are probably due to using the arms, or to irritation from rubbing, but as these results do not appear to occur with humanised lymph it is possible the calf lymph alone may be to blame.

The amount of lymph yielded by each calf, after having undergone the process previously described, varies considerably; I have obtained from one calf sufficient for the vaccination of 4,500 people, the smallest yield supplied material for 752 people, and I generally count on getting sufficient for 2,000 vaccinations from each animal.

The virulence of this lymph is certain, for out of 37,274 primary and re-vaccinations performed in 1890, there were only 2,927 failures.

Other details are essential in carrying out this responsible work; a careful thermometric record of each animal and a description of each should be kept from the day it enters the establishment. The yield of lymph and its distribution should also be noted in order that no difficulty may be experienced in tracing the source of each supply sent out, which should in every case bear the number of the calf and date of collection. These details inspire confidence, and are not to be neglected.

## Conclusion.

The last point I have to deal with is a consideration of the system employed in this country for the instruction of the future members of the veterinary profession in the principles of sanitary science. We have endeavoured to demonstrate the many ways in which veterinary hygiene may assist the State, the public, and the private owner, and have given good reasons for believing that the welfare of our valuable horses and cattle depends entirely on the intelligence exhibited by those responsible for their watering, feeding, stables, ventilation, drainage,

grooming, shoeing, &c., &c., but what measures do we take to ensure the instruction of the profession in these essentials?

I am sorry to say that the Royal College of Veterinary Surgeons requires no instruction to be given in veterinary hygiene; the students are not examined on the subject, and are obviously not taught. This short-sighted policy in a college which has decidedly attempted to raise the standard of veterinary education in this country is greatly to be deplored, especially as such comparatively unimportant collateral sciences as bacteriology and botany are fully dealt with. A man with a smattering of bacteriology is, scientifically speaking, a dangerous person, and the time devoted to the study of these collateral sciences, which can only be successfully employed by specialists, would, if directed to the study of theoretical and practical hygiene, make the future practitioner a stronger and more useful man.

I trust this Congress will assist those of us anxious to impress the Royal College of Veterinary Surgeons with the importance of introducing hygiene into our schools, by supporting a resolution to that effect.

The vital statistics of animals we know nothing about, excepting in the army and some large companies. It is rather difficult to believe that we, the most wealthy horse owning country in the world, have no idea how many horses there are in Britain! Need I say, after this, that we have no knowledge of the births or deaths.

The returns furnished by the Agricultural Department of the Privy Council only deal with agricultural horses. I trust a horse census will be one of those things of the near future, and that this Congress will represent to Her Majesty's Government the necessity of having some knowledge of the horse population of the kingdom.

## DISCUSSION.

**Mr. Goodall, F.R.C.V.S.** (Christchurch), having offered a few remarks with reference to this paper, **The President** said that the programme which the Section had assigned to themselves was now accomplished, and he thanked them very much for their sustained attention during the week, and for their valuable contributions to the debates.

**Professor Chauveau** (Paris), in a felicitous and much applauded speech in French, proposed a vote of thanks to the President of the Section. He said that he was extremely happy to be able to return in time to propose, as a foreign visitor to the Congress, a cordial vote of thanks to their distinguished President. His assiduous attention and active interest in their debates, his charming personality, and the admirable manner in which he had directed and distributed the precious time allotted to the numerous speakers, deserved their most sincere gratitude. He offered his hearty congratulations to the President upon the success of the Section over which he had so ably presided.

**Dr. D. E. Salmon** (Washington, U.S.A.) said that it afforded him great pleasure to endorse what Professor Chauveau had said, and to second the resolution thanking the President for the able and impartial manner in which he had presided over the meetings of the Section.

The motion having been carried by acclamation,

**The President** said he felt exceedingly gratified at having this vote of thanks given to him. What he had done was a very small matter indeed, as compared with the valuable addresses to which they had listened. He ventured to think that they in that Section might honestly congratulate themselves upon the tone and character of their discussions. He had no means of knowing what had been done in the other Sections, but they were agreed that when their papers and discussions had been distributed and digested by people all over the world, great good could not fail to result. Their thanks were due to those gentlemen who had come so far from all parts of Europe and of the world to take part in the Congress, and especially to those who had read such valuable papers.

**Dr. T. W. Hime** (Bradford) said he felt the Section should not disperse without passing a vote of thanks to the officers of the Section, and especially to the three Honorary Secretaries (Mr. Ernest Clarke, Mr. W. Duguid, and Dr. Sims Woodhead). He was sure that his motion would receive the cordial support of all present. Valuable and important as was the Chairman, they could not get on without working members, whose co-operation was indispensable to carry out the details. Every member of the Section would feel that the Secretaries had done their work thoroughly well, and every one had reason to feel grateful for the courtesy and assistance they had rendered to the readers of papers, and to those who took part in the discussions.

**Professor Walley** (Edinburgh) had great pleasure in seconding this resolution which he, as well as Dr. Hime, thought should be formally placed on record.

This resolution having also been carried by acclamation,

**Mr. Duguid** as Senior Secretary, expressed his thanks and those of his two colleagues for the kind manner in which their services had been acknowledged. For his own part, he scarcely deserved any acknowledgment, for most of the work had fallen on his colleague, Mr. Clarke, who had carried out the arrangements both before and during the time of the Congress.

**Mr. Ernest Clarke** having been also called upon to respond, expressed his high gratification at the very complimentary way in which the services of his colleagues and himself had been referred to. He (Mr. Clarke) had been connected with the Section since its earliest formation, and he was proud of his association with what he thought might fairly be described as one of the most practical and most successful departments of the entire Congress. Sir Nigel Kingscote, in his opening remarks, had referred to their gathering as being the first assemblage of medical men, veterinarians, and agriculturists that had ever met on common ground to discuss questions of the profoundest importance to the world at large. He thought that, in view of the conspicuous success of the Section, and the sustained interest taken in its deliberations from beginning to end, it would be safe to predict that it would not be the last. He hoped that at some future Congress, when he himself would be in a position of "greater freedom and less responsibility," he might have the happiness of renewing the many pleasant friendships that he had formed

during the past week. Whether that were so or not, he should always entertain most agreeable memories of the Congress; and he could assure them that for any work and anxiety he might have had in the organisation of the Section, he was more than repaid by its conspicuous success and their generous appreciation of the services he had been only too happy to render.

**The President** said that before separating he was sure the Section would wish him to convey to the Geological Society their thanks for permission to hold their meetings in the room in which they were assembled, and for the facilities afforded to the Section by that Society. Thanks were also due to the authorities of the Royal Veterinary College for their loan of the lantern and other apparatus used for the demonstrations, and for the many interesting microscopic objects which had been on view during the week. In taking leave of them, and declaring the Section dissolved, he had only once more to thank the Vice-Presidents, the Council and the Secretaries of the Section for the valuable assistance which they had rendered him; to the authors of the very interesting papers read before the Section; and last but not least, to the gentlemen who, by their attendance there, and by their contributions to the Debates, had made the Section so remarkable a success.

The Section then dissolved.

# INDEX.

## A.


Page

Abattoirs, general provision of public - - - - 179
Achorion Quinckeanum, Zopf. (Sy. A. Arloingi) - - - 88
—— Schoenleini, Remak - - 89
Actinomyces bovis, Harz - - 92
Actinomycose chez l'Homme, Trois cas d'. (Drs. Doyen et Roussel) 197
Actinomycosis. (Prof. Crookshank) - - - - 192
—— in man - - - - 196
——, manifestations of - - 193
——, prevalence of, in England - 193
—— ——, in Australia - - 194
—— ——, in the United States - 195
Adams (Matthew A.), remarks on The Inspection of Meat with regard to Prevention of Disease - 186
Air, importance of pure, to health of animals - - - 227
Ankylostoma duodenalis, Sur l'enkystement des larves mûres de l' - - - - 191
Anthrax and its relations to workers in various trades (W. Duguid) - - - 211
Anthrax, different forms of - 215
——, discovery of the bacillus anthracis - - - 213
——, disposal of carcasses of animals which have died of - 217
——, history of - - - 212
——, geographical distribution of - 212
——, occupations in which infection most frequently occurs - - 215
——, prevention of - - - 215
—— ——, in man - - 218
——, transmission of - - 214
——, varying susceptibility of different kinds of animals to - 213
Armstrong (Dr. Henry E.), remarks on Infectious Udder Diseases of the Cow - - - 150
—— ——, remarks on dairy sanitation - - - - 169
Aspergillus fumigatus, Fresenius - 96


## B.


Page

Babes (Prof. V.), Études sur la rage et sur la vaccination antirabique - - - 22
Ballard (Edward, M.D.), On meat infections, food poisoning - 102
Bardach (Dr. J.) Résultats statistiques de l'institut antirabique d'Odessa; 18 tableaux - - 35
Barking, outbreak of diptheria in - 146
Barrett (W. F.), remarks on Actinomycosis - - - - 202
"Beri-beri," disease known as - 120
Blackwell (Dr. Elizabeth), remarks in discussion on rabies - - 55
Blanchard (Dr. R.), remarks on the infection of meat and milk - 127
—— ——. Sur les Végétaux parasites (non-microbiens) transmissibles des animaux à l'homme et réciproquement - - 88
Bordoni-Uffreduzzi (Dr.) Sur le traitement antirabique de Pasteur - - - 42
Botryomyces - - - 97
Brown (Prof., C.B.), remarks on Végétaux parasites transmissibles des animaux à l'homme, etc. - - - - 100
—— ——, remarks on rabies - 56
—— ——, remarks on anthrax and its relations to workers in various trades - - - 220
—— ——, remarks on infectious udder diseases of the cow - 153
—— ——, remarks on dairy sanitation - - - - 167
—— ——, remarks on the inspection of meat with regard to prevention of disease - - 187
—— ——, remarks on the infection of meat and milk - - 129
Budapest, ueber die praktischen Erfolge der antirabischen Schutzimpfungen in, wahrend des

Page

ersten, vom 15 April 1890—14 April 1891, sich erstreckenden Jahres. (A. Högyes.) - - 30
Bujwid (Dr. O.). Résultats des vaccinations antirabiques obtenus à Varsovie - - - 51

## C.

Carlisle food poisoning case. (A.) 108
—— —— (B.) - - - 110
Carsten (Dr. B.). Trichinosis in the Netherlands - - - 56
Cercomonas intestinalis de l'homme et des animaux, sur la diffusion des - - - - 190
Charbon (Anthrax). Sur les dangers que le, fait courir aux ouvriers des différents corps dé metiers. (Prof. A. Chauveau) 203
—— ——, traitment des dèpouilles à l'usine - - - - 208
—— ——, traitement des dépouilles aux lieux de production - - 207
—— ——, travail des cornes - 209
—— ——, travail des crins et des laines - - - - 208
—— ——, travail des peaux - 209
——, mesures propres à faire disparaître ou diminuer les cas de, sur les animaux - - 204
Chauveau (Prof.) remarks on infectious udder diseases of the cow - - - - 154
—— ——, remarks on concluding resolutions - - - 246
—— ——, sur les dangers que le Charbon (anthrax) fait courir aux ouvriers des différents corps de métiers - - - - 203
Chester food-poisoning case - 106
Chioniphe Carteri - - - 97
Clarke (Ernest) remarks in reply to vote of thanks - - 247
Collins (Dr. W. J.). Remarks on infectious udder diseases of the cow - - - - 151
Cow, infectious udder diseases of, in relation to epidemic diseases in the human subject. (E. Klein, M.D.) - - - 130
Crookshank (Prof.) Actinomycosis 192
—— ——, remarks on actinomycosis - - - - 203
—— ——, remarks on infectious udder diseases of the cow - 148

## D.

Page

Dangers que le charbon (anthrax) fait courir aux ouvriers des différents corps de métiers. (Prof. M. A. Chauveau) - 203
Différentes méthodes de vaccination antirabique - - 26
Diphtheria, dissemination of, by milk - - - - 145
Diseased meat, the appointment of competent assessors to sit with magistrates and assist them, when necessary, in the hearing of cases relating to - 183
Diseases of animals in Great Britain, table showing number of cases of contagious - - 240
Dog-tax, necessity for imposition of, and of registration - - 21
Doyen et Roussel (Drs.), Trois cas d'actinomycose chez l'homme - 197
Drysdale (Dr. C. R.), remarks in discussion on rabies - - 54
Duguid (W.), Anthrax and its relations to Workers in various Trades - - - 211
—— ——, remarks on vote of thanks - - - 247

## E.

Edinburgh disease, the, in milch cows - - - - 144
Études sur la rage et sur la vaccination antirabique. (V. Babes) - - - 22

## F.

Finkelstein (Dr. T.), Résultats statistiques de l'Institut Antirabique de Tiflis - - - 47
Fleming (George, C. B., LL.D.), remarks on actinomycosis - 203
—— ——, remarks on anthrax - 219
—— ——, remarks on dairy sanitation - - - 170
—— ——, remarks on the inspection of meat with regard to prevention of disease - - 186
—— ——, remarks on the infection of meat and milk - - 128
—— ——, The propagation and prevention of rabies - - 16
Food, importance of, in maintaining the health of animals - 229

Page

Food-poisoning, the Carlisle case. (A.) - - - 108
—— ——, the Carlisle case. (B.) 110
—— ——, the Chester case - 106
—— ——, the Iron Bridge case - 108
—— ——, the Middlesbrough pneumonia epidemic - - 112
—— ——, the Nottingham series of cases - - - - 105
—— ——, the Oldham case - 106
Food-poisoning, On meat infections: (Dr. E. Ballard) - 102
Food-poisoning, the Portsmouth case - - - - 111
—— ——, the Retford case - 109
—— ——, the Welbeck epidemic - 105
—— ——, the Whitchurch brawn case - - - 107
—— ——, the Whitchurch roast pork case - - - - 107
—— ——, the Wolverhampton tinned salmon case - - 107

## G.

Gale sarcoptique de la chèvre - 64
—— ——, du chat - - 67
—— —— du cheval - - 63
—— —— du chien - - - 66
—— —— du dromadaire - - 65
—— —— du lama - - - 65
—— —— du lion - - - 66
—— —— du mouton - - 64
—— —— du porc - - - 65
—— —— du wombat - - 66
Germes charbonneux contenus dans les matières premières utilisées dans l'industrie, mesures propres à détruire les - - - 206
Goodall (Thomas B.), remarks on actinomycosis - - - 201
—— ——, on Veterinary Hygiene - 246

## H.

Hendon and Wiltshire cow-diseases, differences between - - 142
Hendon cow disease - - - 132
Hendon, outbreak of scarlatina at - 130
Hill (Dr. A. Bostock), remarks on infectious udder diseases of the cow - - - - 153
Hime (Dr. T. W.), remarks on rabies - - - - 58-6
—— ——, remarks on anthrax - 219
—— ——, remarks on infectious udder diseases of the cow - - 152
—— ——, vote of thanks to the honorary secretaries - - 247
Högyes (Prof. Dr. A.). Ueber die praktischen Erfolge der antirabischen Schutzimpfungen in Budapest wahrend des ersten, vom 15. April 890—14 April 1891, sich erstreckenden Jahres - 30
Hunting (W.), remarks on infectious udder diseases of the cow - 150
Hydrophobia, table giving results of inoculation in the years 1886–1890 - - - - 12

## I.

Infection of meat and milk. (V. C. Vaughan) - - - 118
—— ——, causes of - - - 118
—— ——, with saprophytic, toxicogenic germs - - - 121
Infections: On meat, food poisoning. (Dr. E. Ballard) - - - 102
Infectious udder diseases in the cow in relation to epidemic diseases in the human subject. (E. Klein) - - - 130
Inspection of animals and meat to be used for the food of man, general systematic - - - 182
Inspection of meat with reference to the spread of disease. (D. E. Salmon) - - - - 171
Inspection of meat with regard to the prevention of diseases. (Francis Vacher) - - 177
Inspectors of meat, the appointment of competent - - - 181
Ivanof (M. Serge), remarks on actinomycosis - - - 200

## K.

"Kakke," disease known as - - 120
Kingscote (Sir Nigel), K.C.B., Presidential address - - 7
——, remarks on concluding resolutions - - - 246–8
——, remarks on infectious udder diseases of the cow - - 154
—— ——, remarks on dairy sanitation - - - - 171

Page

Kraïouchkine (Dr. V.). Les Résultats des vaccinations antirabiques à St. Pétersbourg du 1–13 Juillet 1886 au 1–13 Janvier 1891 46
Klein (E., M.D.). Infectious udder diseases of the cow in relation to epidemic diseases in the human subject - - - - 130

## L.

Lepocolla repens, Eklund - - 94
Licensing of all butchers and their premises - - - 179
Lodge (Dr. Samuel), remarks on anthrax - - - - 218

## M.

Maladies infectieuses des animaux qui peuvent être transmises à l'homme et réciproquement. (Prof. E. Perroncito) - - 189
McFadyean (Prof.), remarks on actinomycosis - - - 202
—— ——, remarks on infectious udder diseases of the cow - - 149
—— ——, remarks on the inspection of meat with regard to prevention of diseases - - 187
Meat, The inspection of, with reference to the spread of disease. (D. E. Salmon) - - - 171
Meat infections, food poisoning. (Dr. E. Ballard) - - 102
Meat, the inspection of, with regard to the prevention of disease. (Francis Vacher) - - - 177
Microphytes: Aspergillus fumigatus, Fresenius - - - 96
——, Microsporon anomaron, Vidal 98
——, —— Audouini, Gruby - 93
——, —— furfur, Ch., Robin - 96
——, —— minutissimum - - 98
——, —— ovale, Bizzozero - - 98
——, —— porophyton, Megnin - 97
——, —— trachomatosum, Noiszewski - - - - 98
——, —— Monilia sputrivia, Gaillipp - - - - 98
Middlesbrough pneumonia epidemic 112
Milchversorgung mit Hinsicht auf uebertragbare Krankheiten, Die Regelung der. (Prof. Ostertag) 155
Milk and meat infection. (V. C. Vaughan) - - - 113
Milk, importance of infection of - 126
——, difficulty of sterilization of - 127

## N.

Page

Nocard (Prof.), remarks on rabies 53
—— ——, remarks on actinomycosis - - - - 199
Nottingham food poisoning cases - 105

## O.

Odessa, Résultats statistiques de l'Institut antirabique d', (J. Bardach) - - - 35
Officers, list of - - - 3
Oidium albicans, Ch. Robin - 98
—— lactis - - - - 98
Oldham food poisoning case - 106
Ostertag (Prof. Dr.). Die Regelung der Milchversorgung mit Hinsicht auf uebertragbare Krankheiten - - - - 155
—— ——, remarks on rabies - 55
—— ——, remarks on infectious udder diseases of the cow - 149
—— ——, remarks on the inspection of meat with regard to prevention of disease - - 185

## P.

Parasites Champignons de l'homme 88
—— Achorion Schoenleini, Remak 89
—— Trieophyton Depilans, Mégnin 90
—— ——, Tonsurans, Malmsten - 91
Parasites Externes - - 58
—— —— Stationnaires - - 61
—— ——, Permanents, Chorioptes 67
—— ——, Demodex folliculorum - 68
—— ——, Psoroptes - - 67
—— ——, Sarcoptes minor - 66
—— ——, Sarcoptes mutans - 67
—— ——, Sarcoptes scabiei - 63–6
—— ——, stationnaires - - 61
—— ——, Périodiques, Dermatobia 62
—— ——, Hypoderma - - 62
—— ——, Ixodes ricinus, etc. - 62
—— ——, Oehromyia - - 61
—— ——, Sarcophaga magnifica - 61
—— ——, Sarcopsylla - - 62
—— ——, Trombidium (Bruyet) - 62
Parasites externes temporaires - 58
—— —— Argas - - - 60
—— —— Dermanyssus - - 59
—— —— Glossina - - - 202
—— —— Hippobosca - - 59
—— —— Pulex - - - - 59
—— —— Stomoxys - - 59
—— —— Simulium - - - 59
—— —— Tabanidæ - - 59

Page
Parasites internes - - - 68
—— —— Ascaris mystax - 77
—— —— Balantidium coli - 71
—— —— Bothriocephalus latus - 82
—— —— Coccidium oviforme; perfor.; bigeminum - - 69
—— —— Distoma hepaticum; lanceolatum - - - 73
—— —— D. sinense - - - 75
—— —— D. truncatum - - 74
—— —— Echinococcus - - 80
—— —— Echinorhynchus hominis 76
—— —— E. moniliformis - 76
—— —— Eustrongylus Gigas - 77
—— —— Filaria medinensis - 78
—— —— Hæmopis - - 79
—— —— Lamblia intestinalis - 70
—— —— Linguatula - - 79
—— —— Mesogonimus Westermanni - - - 75
—— —— Oxyuris vermicularis - 77
—— —— Strongylus paradoxus - 78
—— —— Tænia canina - - 72
—— —— T. saginata - - 82
—— —— T. serrata - - - 72
—— —— T. solium - - 81
—— —— Trichina spiralis - 83
Parasites internes passant des animaux à l'homme par transmission immédiate - - - 80
Parasites internes passant des animaux à l'homme par transmission médiate - - - 69
Parasites (non-microbiens) transmissibles des Animaux à l'Homme et réciproquement, Sur les Végétaux (R. Blanchard) - 88
Parasites stationnaires permanents 63
Parasites transmissibles des Animaux à l'homme, envisagés spécialement au point de vue de la Prophylaxie. (Prof. Railliet) 57
Pearson (Leonard) remarks on dairy sanitation - - - 166
Perroncito (Prof. E.). Maladies Infectieuses des Animaux qui peuvent être transmises à l'homme, et réciproquement - 189
—— ——, remarks on végétaux parasites transmissibles des animaux à l'homme, etc. - - 101
—— ——, remarks on dairy sanitation - - - 168
Ponfick (Prof.), remarks on actinomycosis - - - - 199
Portsmouth food poisoning case - 111

Page
Presidential address (Sir Nigel Kingscote, K.C.B.) - - 7
Preuves pour l'efficacité du traitement antirabique - - 28
Prevention de la Rage après morsure. Résultats obtenus à l'Institut Pasteur au moyen des inoculations anti - rabiques. (Dr. E. Roux) - - - 8
Propagation and prevention of rabies. (Dr. George Fleming) - 16
Propagation du virus rabique - 25
Proteus virulentissimus - - 189

## R.

Rabid dogs, necessity for destruction of - - - - - 17
Rabies, influence of wandering dogs in the propagation of - 18
——, propagation and prevention of. (Dr. G. Fleming) - - 16
——, regulations in force in Berlin for dealing with - - - 19
——, table showing number of cases of, in England, during the years 1887–90 - - - 17
——, value of muzzle in suppressing 18
Rage, Etiologie et histologie de la - 22
——, Études sur la, et sur la vaccination antirabique. (Prof. V. Babes) - - - - 22
——, Prévention de la, après morsure.—Résultats obtenus a l'Institut Pasteur àu moyen des inoculations anti-rabiques. (Dr. E. Roux - - - - 8
Railliet (Prof.) Les parasites transmissibles des animaux à l'homme envisagés spécialement au point de vue de la prophylaxie - - 57
Redfern (Prof.) remarks on rabies 55
Regelung der Milchversorgung mit Hinsicht auf uebertragbare Krankheiten. (Prof. Ostertag) - 155
Registration of all butchers and their-premises - - - 179
Résultats des vaccinations antirabiques à St. Pétersbourg, du 1–13 Juillet 1886 au 1–13 Janvier 1891. (V. Kraïouchkine) - 46
Résultats des Vaccinations Antirabique obtenus à Varsovie. (O. Bujwid) - - - 51
Résultats statistiques de l'Institut antirabique d'Odessa. 18 Tableaux. (J. Bardach) - - - 35

Page
Retford food poisoning case - - 109
Robinson (M. K.), remarks on dairy sanitation - - - 170
Roussel et Doyen (Drs.). Trois Cas d'Actinomycose chez l'Homme - 197
Roux (Dr. E.). Prévention de la rage après morsure. — Résultats obtenus à l'Institut Pasteur au moyen des inoculations antirabiques - - - 8
—— ——, remarks on rabies - 56

## S.

St. Pétersbourg, Résultats des vaccinations antirabiques à, de Juillet 1886 à Janvier 1891. (V. Kraïouchkine) - - 46
Salmon (Dr. D. E.), remarks on actinomycosis - - 200
—— ——, remarks on concluding resolutions - - - 247
—— ——, The inspection of meat with reference to the spread of disease - - - 171
Scarlatina and diseases of the cow - 130
—— ——, extract from the *Practioner* on - - - 135
Selenesporium cuticola, R. Bl. - 97
Sergeant (Edward), remarks on dairy sanitation - - - 171
Simpson (Sir Henry), remarks on rabies - - - - 55
—— ——, remarks on actinomycosis - - - 203
Slaughter-houses, the closing of private - - - 180
Smith (Fred.). Veterinary Hygiene - - - - 221
Stables, importance of improvements in - - - 232
State veterinary hygiene - - 237
Statistique du traitement préventif de la Rage à la Station de Tiflis 50
Substances chimiques produites par le virus rabique - - - 24

## T.

Tiflis, Résultats statistiques de l'Institut antirabique de. (T. Finkelstein) - - - 47
Traitement Antitabique de Pasteur, Sur le. (Dr. Bordoni-Uffreduzzi) 42
—— ——, Preuves pour l'efficacité du - - - -
Trichinosis in the Netherlands. (Dr. B. Carsten) - - - 56
Trichophyton ovoides, Behrend - 98
—— depilans, Mégnin - - 90
—— Tonsurans, Malmstén - - 91
Trois cas d'actinomycose chez l'homme. (Drs. Boyen et Roussel) - - - 197
Turner (Dr. Geo.), remarks on infectious udder diseases of the cow - - - - 151

## U.

Ueber die praktischen enfolge der antirabischen schutzimpfungen in Budapest wahrend des ersten, vom 15 April 1890—14 April 1891 sich erstreckenden Jahres. (A. Högyes) - - 30

## V.

Vaccination antirabique, différentes méthodes de - - - 26
—— —— Études sur la rage et sur la. (Prof. V. Babes) - 22
Vacher (Francis), remarks on dairy sanitation - - - 167
—— ——, The inspection of meat with regard to the prevention of disease - - - 177
Vaughan (Dr. Victor C.). The infection of meat and milk - 118
Végétaux parasites(non-microbiens) transmissibles des animaux à l'homme et réciproquement. (Dr. R. Blanchard) - - 88
Ventilation, importance, of in buildings inhabited by animals - 229
Veterinary hygiene. (Fred. Smith) 221
—— ——, clipping - - 236
—— ——, grooming - - 235
—— ——, individual - - 234
—— ——, special - - - 240
—— ——, State - - - 237
Virus rabique, substances chemiques produites par le - - - 24
—— ——, propagation du - 25
Viandes charbonneuses, alimentation avec des - - - 210

## W.

Page

Walley (Prof.), remarks on actinomycosis - - - 201
—— ——, remarks on infectious udder diseases of the cow - 149
—— ——, remarks on dairy sanitation - - - - 169
—— ——, remarks on vote of thanks - - - 247
—— ——, remarks on the inspection of meat with regard to prevention of disease - - 186
Water, importance of, in the spread or production of disease amongst animals - - - 226
Welbeck food poisoning case - 105
Whitchurch brawn case - - 107
Whitchurch roast pork case - 107
Wiltshire and Hendon cow diseases, differences between - - 143
Wiltshire cow disease - - 142
Wolverhampton tinned salmon poisoning case - - 107
Wool-sorters' disease - - 215

LONDON: Printed by EYRE & SPOTTISWOODE,
Printers to the Queen's most Excellent Majesty.

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on or before the date last stamped below.

I422 International congress
I61 of hygiene and
1891 demography, 7th,
v.3 London. 38705

| NAME | DATE DUE |
| --- | --- |
| | |